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THE URINARY HEPCIDIN AS A NON INVASIVE MARKER FOR IRON DEFICIENCY ANEMIA IN YOUNG ADULT FEMALE IN NORTH INDIAN POPULATION

Submitted by

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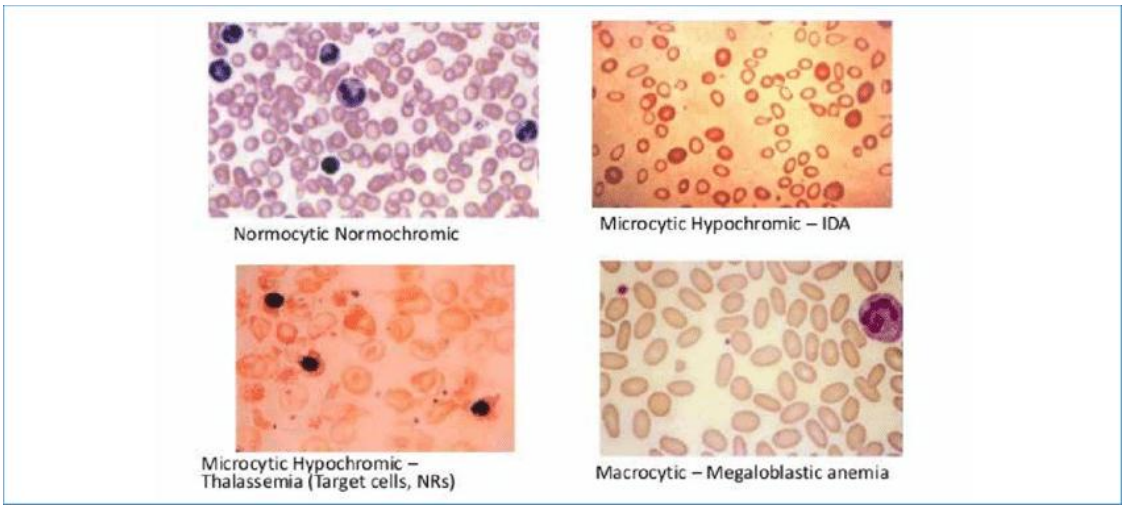
INTRODUCTION

Anemia is a significant global health concern characterized by a reduction in the number of circulating red blood cells (RBCs) or a decrease in hemoglobin concentration below normal physiological thresholds, impairing oxygen transport to body tissues. Hemoglobin, an essential iron-containing protein in RBCs, facilitates oxygen delivery to cells and organs. A decrease in hemoglobin levels compromises the body's oxygen-carrying capacity, resulting in symptoms such as fatigue, weakness, and decreased immunity [1].

The World Health Organization (WHO) defines anemia in non-pregnant women as having hemoglobin levels below 12 g/dL and below 11 g/dL in pregnant women [2]. Anemia has multifactorial etiologies, including nutritional deficiencies, chronic diseases, genetic disorders, and infections. Among these, iron deficiency anemia (IDA) is the most prevalent form, accounting for more than 50% of all anemia cases globally [3] [4].



Global Burden of Anemia: Evidence and Prevalence

Anemia is a prevalent condition affecting nearly 24.8% of the global population, with a higher burden in low- and middle-income countries (LMICs). It disproportionately impacts women of reproductive age, pregnant women, and children under five years of age [5]. South Asia and sub-Saharan Africa bear the highest burden of anemia, with significant disparities in prevalence based on regional and socio-economic factors [6]. Among children under five years of age, the prevalence of anemia remains critically high at 56.5% in LMICs, with

younger children aged 6-35 months being more affected than older ones [7]. Maternal anemia, linked to inadequate nutrition and infection burdens, significantly contributes to adverse pregnancy outcomes, including preterm births, low birth weight, and maternal mortality.

Pregnant women in the third trimester have higher odds of developing anemia compared to those in the first trimester [8]. Infections such as malaria and parasitic diseases, alongside socio-economic disparities and inadequate healthcare, are key determinants of anemia prevalence in LMICs. Iron deficiency remains the leading cause of anemia globally, but its relative contribution varies with infection burden [9]. Interventions targeting high-risk populations, particularly children and pregnant women in regions with high infection burdens, are critical for mitigating anemia's public health impact [10].

Indian Context: Evidence and Insights on Anemia Burden

1. High National Prevalence: Anemia affects 57% of women aged 15–49 years and 67% of children under five in India, with a notable increase compared to previous National Family Health Surveys (NFHS-5). Among adolescent girls aged 15–19 years, the prevalence is alarmingly high at 59%, and it remains a significant concern among pregnant women, with a prevalence of approximately 52% [36].

2. Determinants of High Anemia Prevalence: The key contributing factors include:

    - Inadequate dietary intake: Poor consumption of bioavailable iron from heme sources, combined with the intake of iron absorption inhibitors like tea and phytate-rich cereals.

    - Frequent pregnancies: Closely spaced pregnancies exacerbate iron depletion and anemia risk [33].

    - Infections: High rates of hookworm infestation, malaria, and other parasitic infections contribute significantly to anemia, especially in rural areas [34].

3. Regional Disparities: There is considerable regional variation in anemia prevalence. Northern states like Uttar Pradesh, Bihar, and Rajasthan report higher rates (e.g., 63% in Uttar Pradesh), while southern states like Kerala have significantly lower rates, at

around 38%. This disparity underscores the need for region-specific interventions tailored to the socio-economic and cultural context [35].

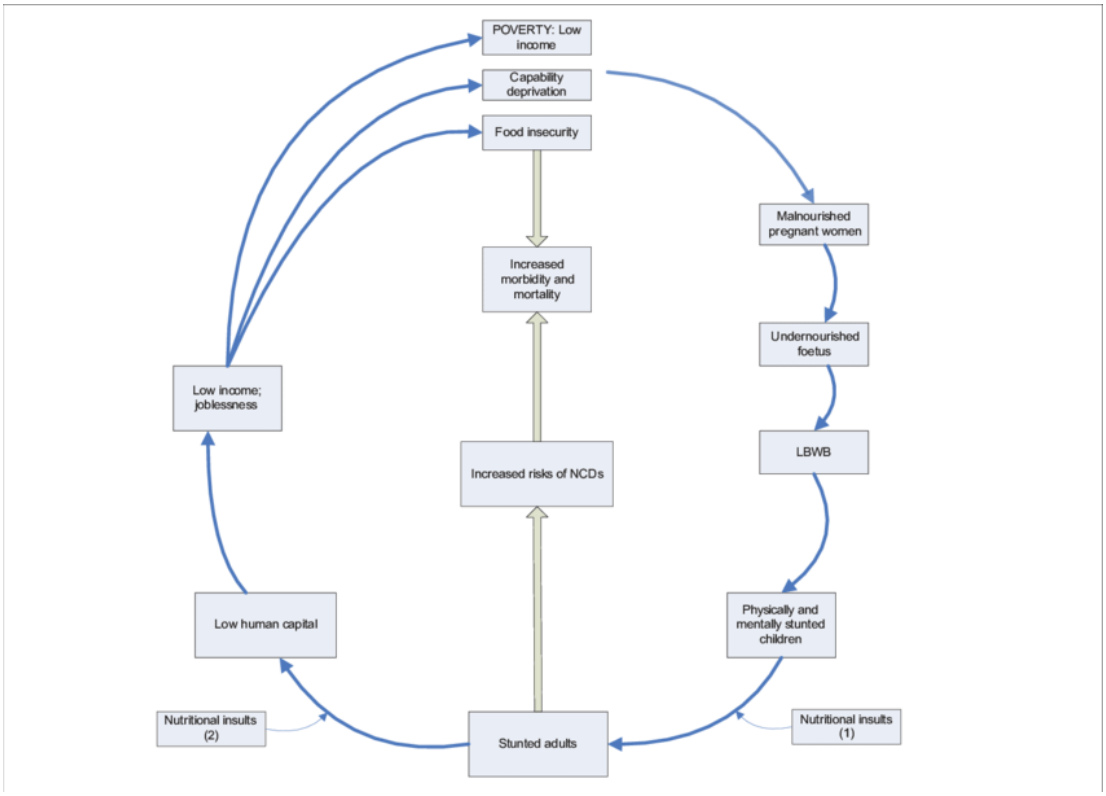
4. Intergenerational Impact: Anemia among Indian women is linked to poor pregnancy outcomes, including low birth weight and stunted growth in children, perpetuating a cycle of malnutrition and poor health outcomes across generations [37].

5. Policy Initiatives and Gaps: The Indian government's Anemia Mukt Bharat (AMB) campaign aims to reduce anemia prevalence through interventions such as:

    - Iron and folic acid supplementation.
    - Deworming initiatives.
    - Public health campaigns to raise awareness about anemia and its prevention [38]. Despite these efforts, challenges persist due to inconsistent program implementation, socio-economic barriers, and insufficient awareness, especially in rural areas [39].

Significance of the Study

Anemia, particularly iron deficiency anemia (IDA), has significant health implications across various stages of life. IDA reduces oxygen transport to tissues, leading to symptoms such as fatigue, weakness, and reduced immunity. In children, iron deficiency during critical periods of growth and development has been associated with impaired cognitive and motor development, resulting in poor academic performance and reduced long-term productivity. Studies indicate that children with untreated IDA score significantly lower on developmental tests compared to their non-anemic counterparts [12]. Furthermore, IDA compromises immune function, increasing susceptibility to infections and prolonging recovery periods [13].



The vicious cycle of malnutrition

In women, particularly those of reproductive age, anemia leads to reduced physical capacity and productivity, impacting their daily activities and economic participation. During pregnancy, untreated IDA increases the risk of adverse maternal and fetal outcomes, such as preterm birth, intrauterine growth restriction (IUGR), low birth weight, and maternal mortality. Severe maternal anemia has been linked to an increased risk of postpartum hemorrhage, sepsis, and maternal death [14]. The World Health Organization (WHO) estimates that anemia contributes to 20% of global maternal deaths [15]. Anemia's compounding effects on maternal and child health emphasize the need for timely diagnosis, effective management, and preventive strategies. Public health interventions aimed at addressing IDA have the potential to significantly reduce maternal and child morbidity and mortality [16].

Socioeconomic Implications of Anemia

The socioeconomic burden of anemia is considerable, especially in low- and middle-income countries (LMICs) like India, where a significant proportion of the population is engaged in labor-intensive occupations. Anemia is known to reduce work productivity by 20%–30%,

resulting in income loss and decreased national economic output [17]. This reduction in productivity has a direct impact on the agricultural sector, where women play a crucial role in farming and food production, ultimately affecting household food security and economic stability [5]. At the national level, anemia increases healthcare costs through frequent hospital visits, medical consultations, and management of severe complications. The World Bank estimates that the economic cost of anemia, including lost productivity and healthcare expenditure, can reduce a country's Gross Domestic Product (GDP) by up to 4% [18]. Beyond direct economic losses, untreated anemia perpetuates a cycle of poverty and poor health. Women with anemia are more likely to give birth to low birth weight infants who face a higher risk of malnutrition, stunting, and cognitive delays, perpetuating the intergenerational cycle of poverty and poor health outcomes [19]. Addressing anemia is therefore not only a public health priority but also an economic necessity, as improving iron status and reducing anemia can lead to better workforce participation, improved national productivity, and sustainable economic growth [19].

Need for Targeted Interventions

Given the multifactorial etiology of anemia and its diverse consequences, a one-size-fits-all approach is insufficient to address this public health issue. Region-specific strategies that consider socio-economic, cultural, and dietary differences are crucial. For instance, in regions with predominantly vegetarian diets, interventions should promote iron-fortified foods and improve non-heme iron bioavailability through dietary modifications [20]. Additionally, in malaria-endemic areas, integrated approaches combining iron supplementation with malaria control measures, such as insecticide-treated bed nets and prophylactic anti-malarial medications, are necessary to prevent exacerbation of anemia [21]. Furthermore, innovative diagnostic tools like urinary hepcidin provide significant potential for improving the early detection and management of iron deficiency anemia (IDA). Unlike traditional serum biomarkers, urinary hepcidin offers a non-invasive, cost-effective alternative for large-scale screening, particularly in resource-limited settings [22]. Incorporating such diagnostic innovations into national health programs could enhance the efficacy of anemia control initiatives, ensuring better health outcomes for vulnerable populations [23].

AIM AND OBJECTIVES

AIM

To Establish Urinary Hepcidin As A Non Invasive Marker For Iron Deficiency Anemia In Young Adult Female In North Indian Population

OBJECTIVES

1. To assess baseline value for urinary hepcidin as a test for iron deficiency anemia
2. To determine iron deficiency anemia based on traditional parameters (serum iron, serum ferritin, total iron binding capacity, complete blood count).
3. Comparison of urinary hepcidin levels with traditional IDA parameters

REVIEW OF LITERATURE

Anemia is a condition characterized by a reduction in the number of red blood cells (RBCs) or a decrease in hemoglobin concentration below normal physiological levels, resulting in impaired oxygen delivery to body tissues. According to the World Health Organization (WHO), anemia affects approximately 1.92 billion people worldwide, with iron deficiency being the most prevalent cause [24]. Anemia can have a significant impact on physical and cognitive performance, immune function, and overall quality of life, particularly in vulnerable populations such as women of reproductive age, pregnant women, and children [25]. The pathophysiology of anemia involves three main mechanisms: reduced RBC production, increased RBC destruction, and blood loss.

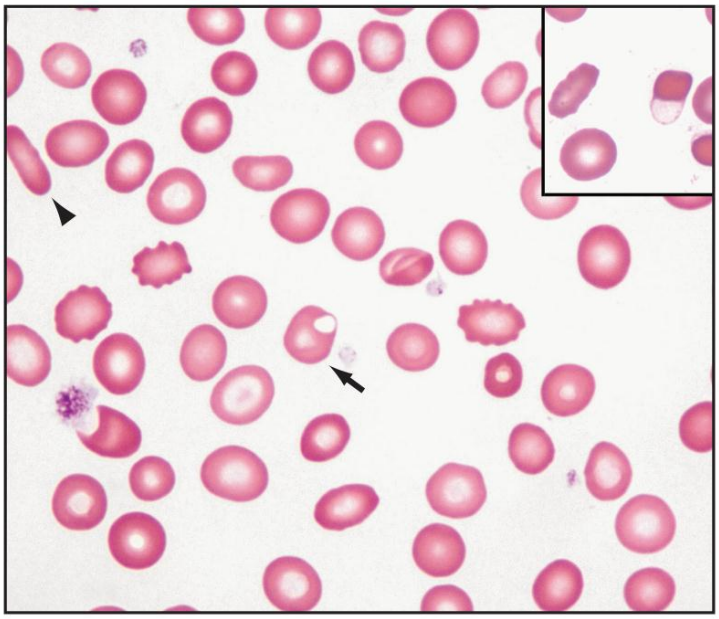


Figure 1: Microcytic Hypochromic Anemia

Iron deficiency anemia (IDA) is the most common form of anemia globally, accounting for more than 50% of cases. It occurs due to insufficient iron levels required for hemoglobin synthesis. Common causes include poor dietary intake of iron, chronic blood loss due to menstruation or gastrointestinal disorders, and increased iron demand during pregnancy [26]. A study conducted by the National Family Health Survey (NFHS) in India reported that 57% of women in the reproductive age group are anemic, highlighting the severity of the problem in low- and middle-income countries [27]. Megaloblastic anemia, another prevalent type, results from vitamin B12 or folate deficiency, leading to defective DNA synthesis and the formation of large, immature RBCs known as megaloblasts. Factors contributing to megaloblastic anemia include malnutrition, malabsorption syndromes, and certain

medications [28]. Hemolytic anemia, characterized by the premature destruction of RBCs, can be either inherited or acquired. Inherited forms include conditions like sickle cell disease and thalassemia, where abnormal hemoglobin synthesis leads to chronic hemolysis. Acquired hemolytic anemia can result from autoimmune diseases, infections, or exposure to specific drugs or toxins. In tropical and subtropical regions, infections such as malaria and hookworm infestation are significant contributors to anemia. These infections either directly destroy RBCs or cause chronic blood loss, exacerbating iron deficiency [24]. Aplastic anemia is a rare but serious form of anemia that occurs due to bone marrow failure. The bone marrow's inability to produce sufficient RBCs, white blood cells, and platelets leads to pancytopenia—a condition characterized by low levels of all blood cells. Causes of aplastic anemia include idiopathic factors, exposure to radiation, certain chemicals, and the use of cytotoxic drugs [29]. Nutritional deficiencies, chronic diseases, infections, and genetic disorders represent the broad categories of anemia etiology. Nutritional anemia, which encompasses iron, folate, and vitamin B12 deficiencies, is the leading cause of anemia worldwide [30]. Chronic diseases such as chronic kidney disease, cancer, and inflammatory conditions contribute to anemia by impairing erythropoiesis and iron utilization. The pathophysiology of anemia involves either insufficient RBC production due to deficient nutrients or bone marrow dysfunction, excessive RBC destruction as seen in hemolytic anemia, or chronic blood loss, which depletes the body’s iron stores [31]. Anemia is a major public health concern due to its significant burden on morbidity and mortality, especially in low-income regions. Effective strategies to address anemia include improving dietary iron intake, implementing iron supplementation programs, and controlling infections [25]. Recent data highlight the urgent need for innovative diagnostic markers, such as urinary hepcidin, to better understand iron homeostasis and provide more effective management of IDA [32].

Global and Indian Proportions of Anemia Prevalence

Comparative Statistics in Global Prevalence of Anemia

The prevalence of anemia varies significantly across regions, genders, and age groups, reflecting differences in dietary patterns, socioeconomic status, and healthcare access. According to the World Health Organization (WHO), anemia affects 29.9% of women of reproductive age globally. The prevalence is highest in South Asia and Sub-Saharan Africa, where over 40% of women are anemic. A gender-based analysis indicates that anemia is more common in females than males due to additional iron losses during menstruation and the increased iron demands of pregnancy [33]. Among children under five, the global prevalence of anemia is estimated at 39.8%, with nutritional deficiencies being the primary cause [54].

In terms of regional variation, high-income countries report a lower prevalence of anemia, often below 10%, owing to better dietary diversity and access to healthcare services. In contrast, low- and middle-income countries, particularly in South Asia, exhibit much higher rates due to poor dietary intake of iron-rich foods and the burden of infectious diseases such as malaria and hookworm infestation [55]. Anemia is a widespread public health problem that affects over 1.6 billion people globally, accounting for nearly 24.8% of the world's population. Women of reproductive age and children under five years are the most vulnerable populations due to increased iron requirements during menstruation, pregnancy, and early growth phases [34]. Sub-Saharan Africa and South Asia exhibit the highest regional prevalence, with anemia rates exceeding 40% in certain demographic groups [35].

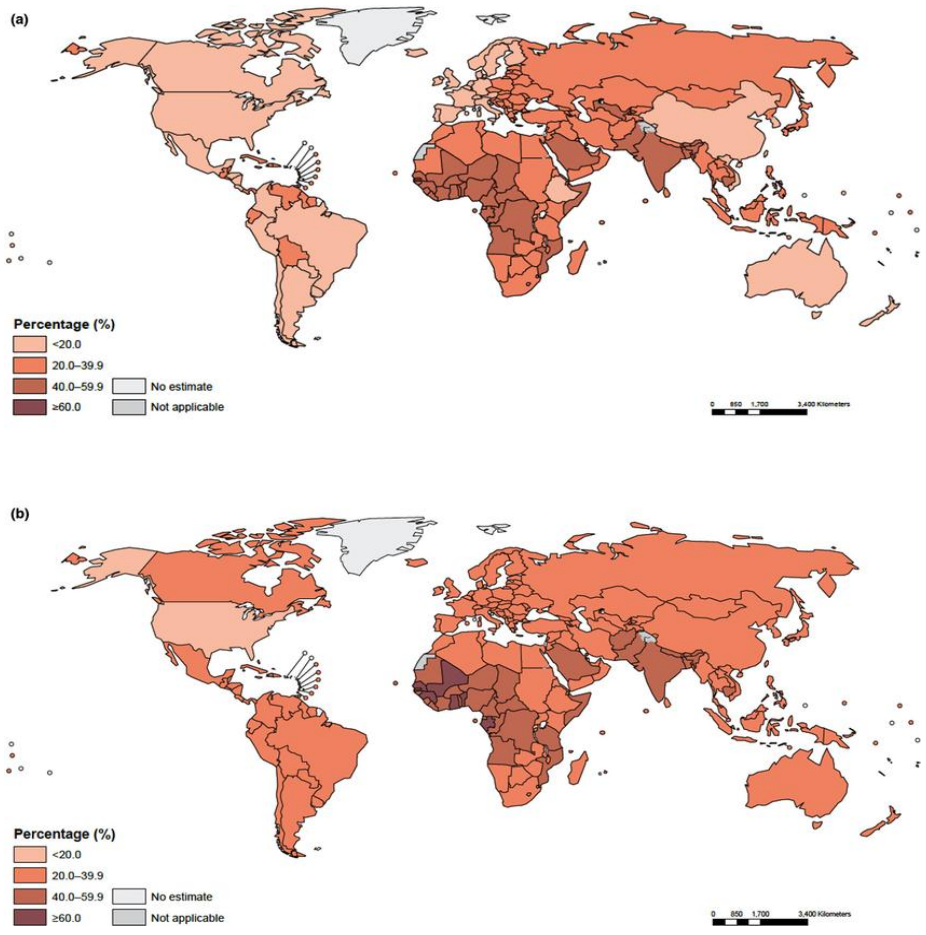


Figure 2: The global prevalence of anemia. (a) depicts the worldwide prevalence of anemia in all women, while (b) demonstrates the prevalence of anemia in those who are pregnant. Iron deficiency is the cause or contributor to at least half the worldwide cases of anemia in reproductive-aged women.

Indian Proportions of Anemia Prevalence

In India, the prevalence of anemia is particularly concerning. The latest data from the National Family Health Survey (NFHS-5) highlights that 57% of women in the reproductive age group (15–49 years) are anemic, reflecting a worsening trend compared to previous surveys [33]. Among adolescent girls aged 15–19 years, the prevalence is alarmingly high at 59%, while pregnant women report a prevalence rate of nearly 52% [36]. Factors such as inadequate dietary iron intake, frequent pregnancies, poor sanitation, and the high prevalence of infections like hookworm and malaria are key contributors to anemia in India. Furthermore, anemia in Indian women not only affects their health but also has intergenerational consequences, contributing to low birth weight, increased infant mortality, and impaired cognitive development in children [37].

Regionally, anemia prevalence shows significant variation within India, with North Indian states like Uttar Pradesh, Bihar, and Rajasthan reporting disproportionately high rates compared to southern and western states. For instance, in Uttar Pradesh, the prevalence of anemia among women of reproductive age is as high as 63%, compared to 38% in Kerala. Socioeconomic factors, dietary habits, and healthcare access disparities contribute to these regional differences [56][57][58].

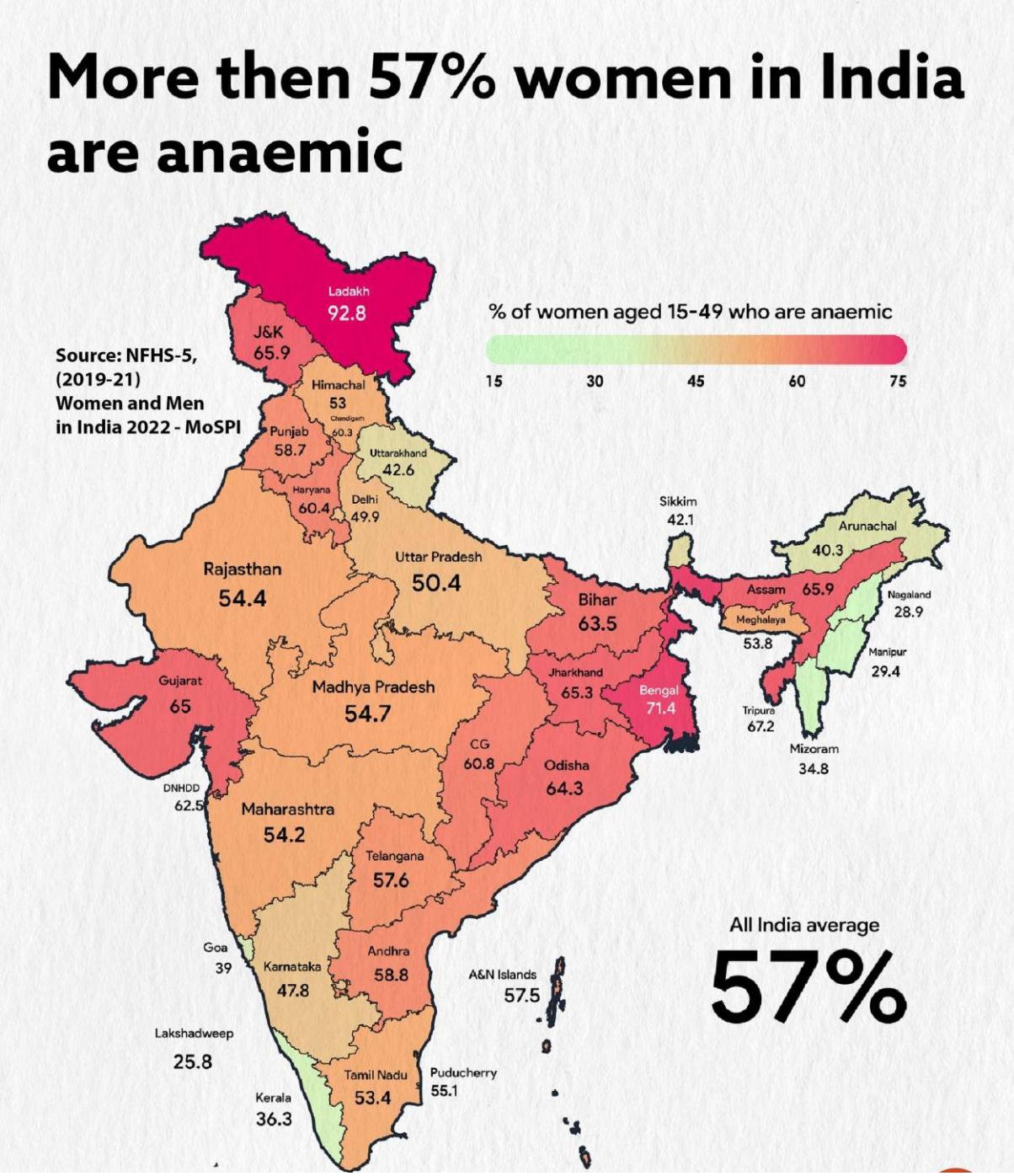


Figure 3: Indian Prevalence of Anemia

Government Initiatives to Combat Anemia in India

The government of India has launched several initiatives, including the Anemia Mukt Bharat (AMB) program, aimed at reducing anemia prevalence by promoting iron and folic acid supplementation, dietary diversification, and deworming interventions. Despite these efforts, anemia continues to pose a significant challenge due to socio-economic disparities, low

awareness, and inconsistent program implementation [38]. The disproportionately high prevalence of anemia in women of reproductive age highlights the urgent need for targeted interventions and innovative diagnostic tools, such as urinary hepcidin, which could improve early detection and management of iron deficiency anemia [39]. Additionally, studies suggest the importance of addressing multiple micronutrient deficiencies, as iron supplementation alone may not fully solve the problem [40].

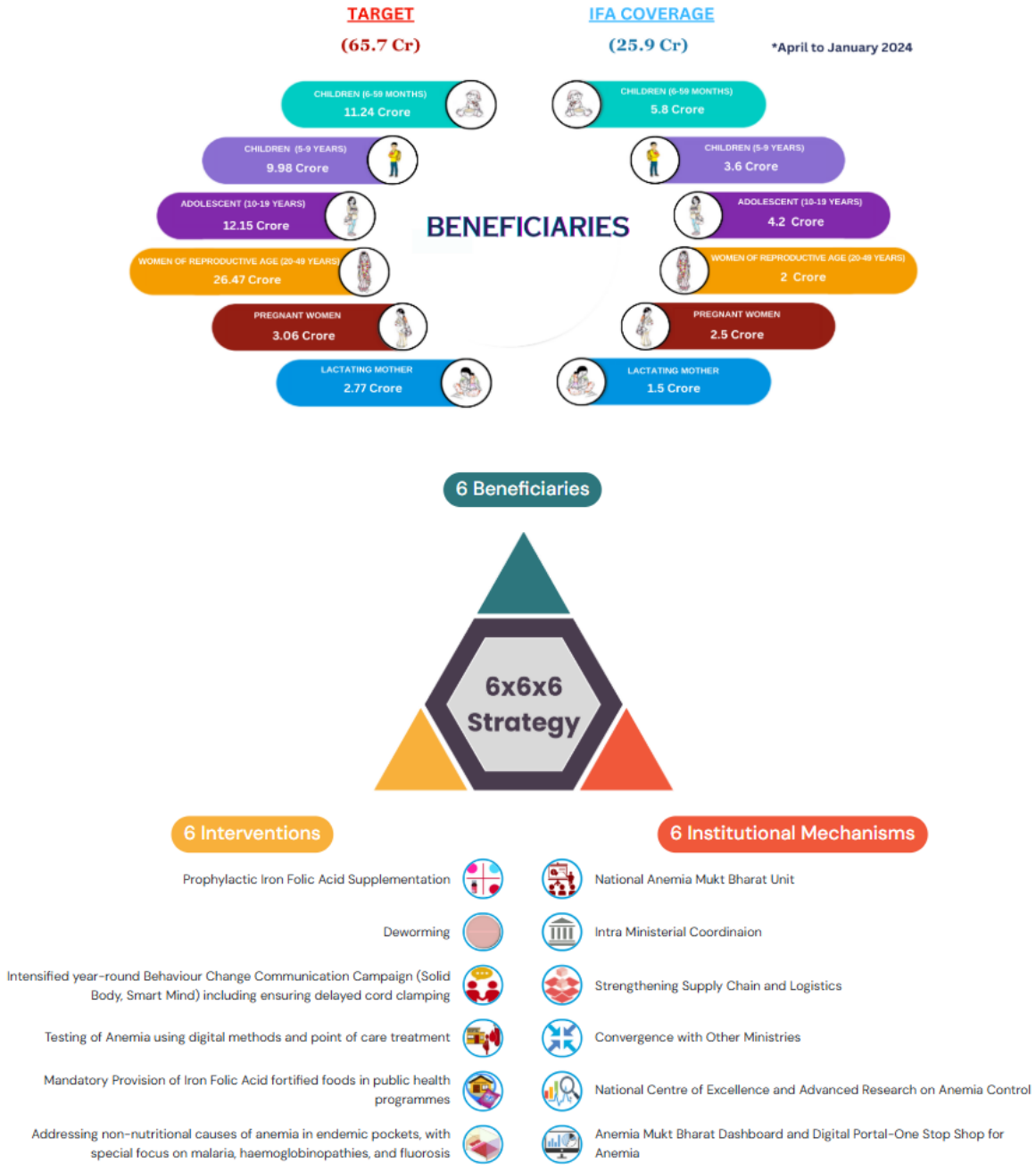


Figure 4 Anemia Mukt Bharat (AMB) program beneficiaries & strategy

Need for Region-Specific Strategies

Given the significant regional disparities in anemia prevalence across India, there is a pressing need for region-specific strategies. These may include enhancing iron supplementation programs, promoting dietary diversification, and addressing socioeconomic determinants of health. The Anemia Mukt Bharat (AMB) initiative, launched by the Indian government, aims to reduce anemia prevalence by promoting iron and folic acid supplementation, deworming, and dietary counseling. However, the success of such programs requires efficient implementation, particularly in high-burden regions like North India [35]. Addressing anemia in young adult females is particularly important, as it directly impacts maternal and child health outcomes, work productivity, and the overall well-being of future generations. Innovative approaches, such as the use of urinary hepcidin as a diagnostic tool, could further improve early detection and management of iron deficiency anemia, especially in resource-limited settings [60].

Iron Deficiency Anemia (IDA) and Its Causes in Women

Iron Deficiency Anemia (IDA) is a specific type of anemia that occurs due to insufficient iron availability in the body, resulting in inadequate hemoglobin synthesis. Hemoglobin, an iron-containing protein in red blood cells, is essential for oxygen transport. When iron levels are insufficient, hemoglobin production decreases, leading to reduced oxygen-carrying capacity of the blood. IDA is the most prevalent form of anemia worldwide, accounting for 50%–60% of all anemia cases, according to WHO reports [28]. The progression of IDA involves three stages: depletion of iron stores, iron-restricted erythropoiesis, and iron deficiency anemia, characterized by low hemoglobin levels and microcytic, hypochromic red blood cells.

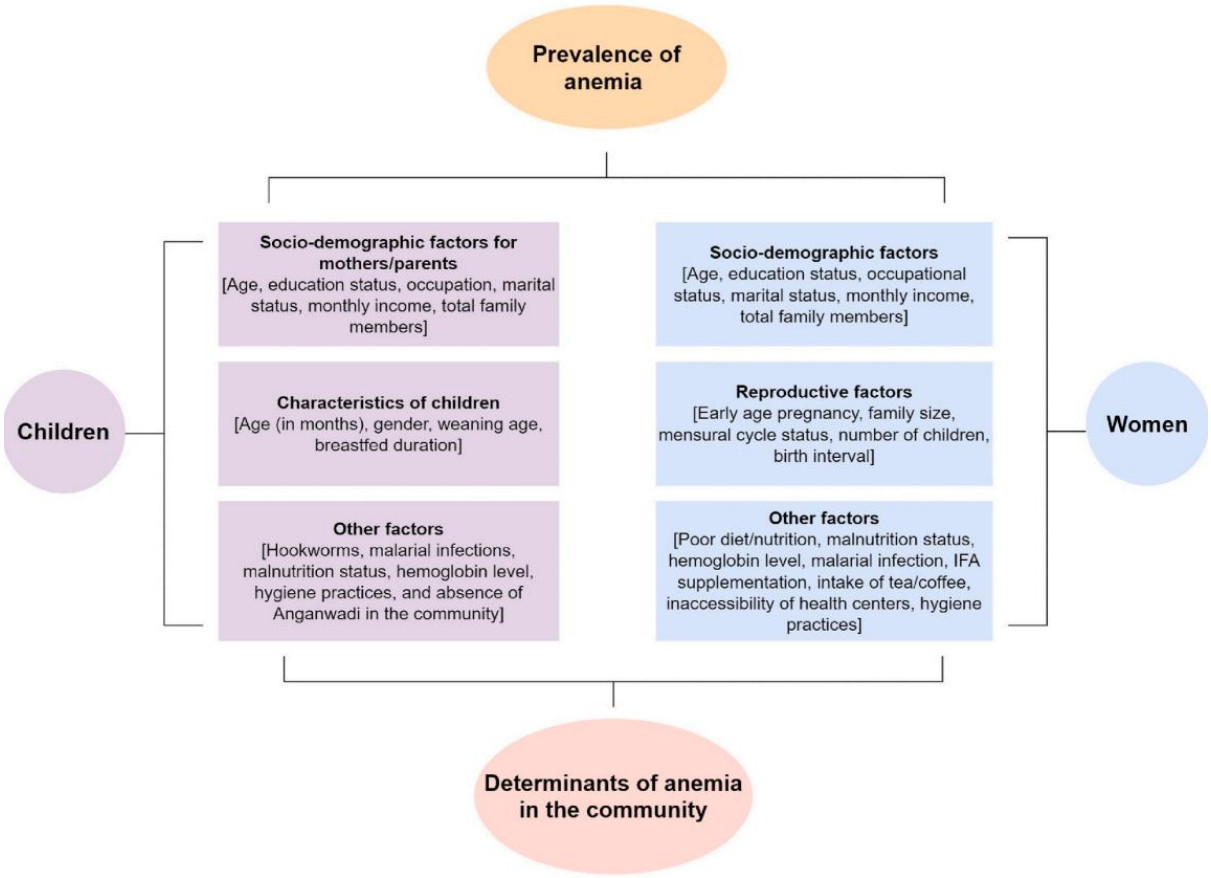

Prevalence of anemia
Children
Socio-demographic factors for mothers/parents
[Age, education status, occupation, marital status, monthly income, total family members]
Characteristics of children
[Age (in months), gender, weaning age, breastfed duration]
Other factors
[Hookworms, malarial infections, malnutrition status, hemoglobin level, hygiene practices, and absence of Anganwadi in the community]
Women
Socio-demographic factors
[Age, education status, occupational status, marital status, monthly income, total family members]
Reproductive factors
[Early age pregnancy, family size, mensural cycle status, number of children, birth interval]
Other factors
[Poor diet/nutrition, malnutrition status, hemoglobin level, malarial infection, IFA supplementation, intake of tea/coffee, inaccessibility of health centers, hygiene practices]
Determinants of anemia in the community

Figure 5 Conceptual framework of the determinants responsible for the prevalence of anemia among children and women.

Primary Causes of IDA in Women

Women are particularly susceptible to IDA due to various physiological and lifestyle factors. The primary causes contributing to IDA in women include:

1. Menstrual Blood Loss

Regular menstrual bleeding is one of the leading causes of iron loss in women of reproductive age. Each menstrual cycle results in an average loss of 20–30 mg of iron, and in cases of heavy menstrual bleeding (menorrhagia), the loss can be significantly higher [41]. Studies have shown that 20%–30% of premenopausal women suffer from iron deficiency due to menstrual blood loss. Women with prolonged or heavy menstruation are at an even higher risk of developing IDA, particularly if dietary iron intake is insufficient to compensate for these losses.

2. Poor Dietary Iron Intake

Low consumption of dietary iron, particularly heme iron, is a major contributor to IDA. Heme iron, found in animal-based foods such as red meat, poultry, and fish, is more bioavailable than non-heme iron from plant sources. In populations with predominantly vegetarian diets, such as in many parts of India, iron absorption is further hindered by dietary inhibitors like phytates, found in cereals and legumes, which reduce iron bioavailability [42]. According to NFHS-5 data, a significant proportion of Indian women do not meet the daily recommended intake of dietary iron, contributing to the high prevalence of IDA [43].

3. Increased Iron Demand During Pregnancy

Pregnancy significantly increases iron requirements to support fetal growth, placental development, and increased maternal blood volume. The daily iron requirement during pregnancy can rise from 18 mg to 27 mg per day. Without adequate supplementation, pregnant women are at high risk of developing IDA, which can lead to adverse maternal and fetal outcomes, including preterm delivery, low birth weight, and increased perinatal mortality [44]. WHO estimates that 40% of pregnant women globally suffer from anemia, with iron deficiency being the primary cause.

Additional Contributing Factors

In addition to the primary causes, several other factors can exacerbate the risk of IDA in women:

- **Frequent Pregnancies**: Closely spaced pregnancies do not allow sufficient time for the body to replenish iron stores, increasing the risk of IDA [45].
- **Lactation:** Postpartum women require additional iron to support breastfeeding, further depleting iron stores if intake is inadequate.
- **Poor Socioeconomic Conditions**: Limited access to iron-rich foods and healthcare facilities increases the prevalence of IDA in low-income populations [46].

Public Health Significance

IDA in women poses a significant public health challenge due to its impact on reproductive health, productivity, and quality of life. The consequences of untreated IDA include reduced physical and cognitive performance, increased susceptibility to infections, and poor pregnancy outcomes. Addressing the causes of IDA in women requires a comprehensive approach involving iron supplementation, dietary improvement, and awareness programs aimed at reducing menstrual-related blood loss and meeting increased iron demands during pregnancy [47].

Consequences of Untreated Iron Deficiency Anemia (IDA)

Physiological Consequences of Untreated IDA

Untreated iron deficiency anemia (IDA) can have widespread physiological impacts due to the body's inability to produce adequate hemoglobin, which is essential for oxygen transport. The reduced oxygen-carrying capacity of blood affects multiple organ systems, leading to the following consequences:

a) Impaired Cognitive Performance

Iron plays a crucial role in brain function, including neurotransmitter synthesis and myelination. Iron deficiency, particularly during critical periods of growth and development, can impair cognitive performance, concentration, and memory. Studies have shown that children and adolescents with untreated IDA score significantly lower on cognitive and psychomotor tests compared to their non-anemic peers. In adults, untreated IDA is associated with reduced work productivity and impaired executive functioning [48].

b) Reduced Physical Capacity

Since hemoglobin is essential for oxygen transport to muscles, iron deficiency leads to decreased endurance and exercise capacity. Individuals with untreated IDA often experience fatigue, weakness, and breathlessness even during mild physical activity. In severe cases, it can cause reduced physical performance, limiting daily activities and work productivity [49].

c) Delayed Growth and Development in Children

Iron deficiency during infancy and early childhood can result in delayed growth and development. It is a critical nutrient for cellular metabolism and growth, and its deficiency can lead to stunted growth, impaired immune function, and increased susceptibility to infections. According to WHO, IDA in children under five years is associated with a 20% higher risk of developmental delays [50].

d) Maternal and Neonatal Health Consequences

Untreated IDA in women of reproductive age, especially during pregnancy, poses significant risks to both maternal and neonatal health. The increased iron requirements during pregnancy, if unmet, can lead to adverse outcomes such as:

- **Increased Maternal Morbidity and Mortality**

  Severe anemia during pregnancy increases the risk of maternal mortality due to complications such as postpartum hemorrhage and sepsis. Pregnant women with untreated IDA are also at higher risk of developing preeclampsia and infections. WHO reports that anemia contributes to 20% of all maternal deaths globally, highlighting its significant impact on maternal health outcomes [51].

- **Adverse Neonatal Outcomes**

Iron deficiency during pregnancy can result in adverse neonatal outcomes, including preterm birth, low birth weight, and intrauterine growth restriction (IUGR). Low iron availability affects fetal iron stores, leading to neonatal iron deficiency, which in turn increases the risk of anemia during infancy. Furthermore, IDA during pregnancy has been linked to impaired cognitive and motor development in offspring during early childhood [52].

Public Health Impact

The consequences of untreated IDA extend beyond individual health, contributing to a substantial public health burden. In low- and middle-income countries, where the prevalence of IDA is highest, untreated anemia exacerbates socio-economic disparities by impairing the health and productivity of affected populations. Addressing IDA through early detection, appropriate treatment, and preventive measures is crucial for improving overall health outcomes, reducing maternal and child mortality, and enhancing economic productivity [53].

Disproportionate Impact on Young Adult Females

Young adult females in India, particularly in North India, show disproportionately high rates of anemia due to a combination of poor dietary intake, menstrual blood loss, and socioeconomic factors. The dietary patterns in many North Indian communities are heavily plant-based, with limited consumption of bioavailable heme iron from animal sources. Furthermore, inadequate access to healthcare and poor awareness regarding iron supplementation contribute to the high prevalence in these regions [59].

WHO Standards for Iron Deficiency Anemia (IDA) in Women

WHO Diagnostic Criteria for IDA

The World Health Organization (WHO) has established specific diagnostic criteria for anemia based on hemoglobin concentration levels. In non-pregnant women, anemia is diagnosed when hemoglobin levels fall below 12 g/dL, while in pregnant women, the threshold is 11 g/dL. These thresholds are used globally to identify and classify anemia into different severity levels:

- Mild anemia: Hemoglobin levels between 11 g/dL and 11.9 g/dL in non-pregnant women
- Moderate anemia: Hemoglobin levels between 8 g/dL and 10.9 g/dL
- Severe anemia: Hemoglobin levels below 8 g/dL WHO emphasizes that hemoglobin levels alone are not always sufficient for diagnosing iron deficiency anemia (IDA). Additional biomarkers, such as serum ferritin, transferrin saturation, and soluble transferrin receptors, may be required to confirm iron deficiency, particularly in populations where infections and inflammation are prevalent, as these conditions can affect ferritin levels [61]. The WHO defines iron deficiency as serum ferritin levels below 15 µg/L in non-pregnant women and 30 µg/L in pregnant women [62].

Iron Supplementation Guidelines

To address the high global burden of anemia, WHO recommends routine iron and folic acid supplementation for high-risk populations, particularly women of reproductive age, pregnant women, and adolescents. The guidelines specify the following interventions:

Non-Pregnant Women

- Dosage: 30–60 mg of elemental iron and 400 µg of folic acid daily for three months annually in settings with high anemia prevalence.
- Objective: To prevent iron deficiency and improve overall hemoglobin levels [63].

Pregnant Women

- Dosage: 30–60 mg of elemental iron and 400 µg of folic acid daily throughout pregnancy and for six weeks postpartum.
- Objective: To meet the increased iron requirements during pregnancy, reduce the risk of maternal anemia, and improve pregnancy outcomes [64].

Adolescent Girls

- Dosage: Weekly iron and folic acid supplementation with 60 mg of elemental iron and 2.8 mg of folic acid in areas where anemia prevalence exceeds 20%.
- Objective: To address the high risk of IDA in adolescents and improve pre-pregnancy iron stores [65].

Maternal Deaths Due to Anemia During Pregnancy and Childbirth

Anemia is a leading cause of maternal deaths globally, significantly contributing to maternal morbidity and mortality, particularly in low- and middle-income countries. According to the World Health Organization (WHO), anemia directly accounts for approximately 20% of maternal deaths worldwide, primarily due to increased risks of severe postpartum hemorrhage, sepsis, and other life-threatening complications during pregnancy and childbirth [109]. A study conducted across multiple countries found that severe anemia during pregnancy elevates the risk of maternal mortality by 2.4 times compared to non-anemic women. In India, the burden of anemia remains exceptionally high, with 57% of pregnant women affected, as reported by the National Family Health Survey (NFHS-5). This high prevalence correlates with the country's maternal mortality ratio (MMR) of 97 per 100,000 live births. Severe anemia (hemoglobin levels below 7 g/dL) increases the risk of maternal mortality by up to 3.5 times compared to non-anemic women [110]. Anemia also contributes to other complications, including preterm labor, low birth weight, and intrauterine growth restriction (IUGR), further exacerbating neonatal mortality rates [111].

Strategies for Reduction:

To reduce maternal mortality associated with anemia, a comprehensive approach is necessary, focusing on prevention, early detection, and effective management. The following strategies have demonstrated efficacy:

a) Iron and Folic Acid Supplementation

The WHO recommends routine iron and folic acid supplementation during pregnancy. Programs such as India's Anemia Mukt Bharat (AMB) initiative offer free iron and folic acid supplements to pregnant women, accompanied by regular hemoglobin monitoring [112].

b) Dietary Improvement

Improving dietary intake of iron-rich foods, particularly heme iron sources like meat, fish, and poultry, is crucial. Non-heme sources, such as legumes and fortified cereals, can be combined with vitamin C to enhance absorption. Reducing dietary inhibitors like tea and coffee also improves iron bioavailability [113].

c) Fortification of Staple Foods

Food fortification, including the addition of iron and folic acid to wheat flour and rice, has been successfully implemented in several countries. National programs aim to expand such fortification efforts to cover wider populations [114].

d) Deworming and Malaria Control

Deworming during pregnancy helps to reduce chronic blood loss caused by intestinal parasitic infections, which are significant contributors to anemia. In malaria-endemic regions, insecticide-treated bed nets (ITNs) and prophylactic anti-malarial medications are essential [115].

e) Early Screening and Treatment

Regular antenatal check-ups, including hemoglobin testing, enable early detection and timely treatment of anemia. Severe cases should be managed with parenteral iron or blood transfusions when necessary to prevent complications during labor and delivery [116].

f) Public Awareness and Education

Increasing awareness of anemia prevention and treatment among pregnant women and their families is critical. Community health workers play a crucial role in promoting adherence to iron supplementation and educating women on proper nutrition [117].

Iron Deficiency Anemia (IDA) as the Primary Cause of Nutritional Anemia in Young Women

Types of Nutritional Anemia

Nutritional anemia refers to anemia resulting from a deficiency of essential nutrients required for the production of healthy red blood cells. The primary types of nutritional anemia include:

a)Iron Deficiency Anemia (IDA)

IDA is the most prevalent form of nutritional anemia, caused by insufficient dietary iron intake, poor iron absorption, or chronic blood loss. Iron is essential for hemoglobin synthesis, and its deficiency leads to impaired oxygen transport and microcytic, hypochromic red blood cells. Globally, 50%–60% of all anemia cases are attributed to iron deficiency [118].

b)Folate Deficiency Anemia

Folate (vitamin B9) deficiency impairs DNA synthesis and cell division, leading to megaloblastic anemia characterized by large, immature red blood cells. Folate deficiency is particularly prevalent among pregnant women, where increased demand exacerbates the risk [119].

c)Vitamin B12 Deficiency Anemia

Vitamin B12 is crucial for proper red blood cell formation and neurological function. Its deficiency causes megaloblastic anemia and can lead to severe neurological complications. Populations with predominantly vegetarian diets are at higher risk of B12 deficiency due to limited dietary sources [120]. Although folate and vitamin B12 deficiencies contribute to nutritional anemia, iron deficiency remains the leading cause worldwide, particularly among young women.

Impact of nutritional anemia the Reproductive Age Group

Young women in the reproductive age group (15–49 years) are disproportionately affected by nutritional anemia, with iron deficiency being the predominant cause. Several factors contribute to this heightened susceptibility:

Consequences on Health and Productivity

IDA in young women has far-reaching consequences on reproductive health, physical capacity, and productivity:

a) Reduced Work Capacity

IDA decreases physical endurance and cognitive performance, leading to reduced productivity. Studies indicate that women with anemia have a 20%–30% lower work output compared to non-anemic women [126].

b) Impaired Reproductive Health

Anemia during pregnancy increases the risk of complications such as preterm labor and low birth weight, further exacerbating neonatal morbidity and mortality [127].

c) Poor Quality of Life

Fatigue, weakness, and frequent infections associated with IDA impair overall well-being, limiting educational attainment and economic opportunities for young women [128].

Regional Studies in North India

Several regional studies have underscored the high prevalence of IDA in North India. In Uttar Pradesh, more than 65% of adolescent girls were found to be anemic, primarily due to dietary inadequacies and socio-economic barriers [131]. Similar studies conducted in Rajasthan and Bihar identified poor healthcare infrastructure and low dietary diversity as critical factors contributing to the high burden of IDA [132]. These findings highlight the urgent need for targeted anemia control programs focusing on improving dietary practices, iron supplementation, and public health education.

Public Health Approach

In addition to individual supplementation, WHO emphasizes the need for a public health approach to anemia prevention and control, which includes:

1. Fortification of staple foods with iron and folic acid, such as wheat flour and rice.
2. Dietary diversification to increase the intake of bioavailable iron through heme-rich foods and iron absorption enhancers like vitamin C.
3. Infection control measures, including deworming programs and malaria prevention, to reduce anemia caused by parasitic infections.
4. Health education campaigns to raise awareness about the importance of iron-rich diets and adherence to supplementation programs [66].

Global Implementation

The WHO's guidelines are being implemented in several countries through national health programs aimed at reducing anemia prevalence. For example, India's Anemia Mukt Bharat (AMB) initiative aligns with WHO's recommendations by providing iron and folic acid supplements to adolescent girls, pregnant women, and lactating mothers. However, despite these efforts, adherence to supplementation programs remains a challenge in many regions due to factors such as side effects, poor access to healthcare, and cultural beliefs [67].

Adopting innovative strategies, such as the use of non-invasive biomarkers like urinary hepcidin, could improve early detection and monitoring of IDA, ensuring timely intervention and better outcomes. WHO continues to advocate for research and development of new diagnostic tools and treatment approaches to enhance anemia management globally [68].

Iron Deficiency Anemia (IDA)

Definition and Classification

Iron Deficiency Anemia (IDA) is defined as anemia caused by insufficient iron to meet the body's requirements for hemoglobin synthesis. The World Health Organization (WHO) classifies IDA based on hemoglobin levels as follows:

- Mild Anemia: Hemoglobin levels between 11 g/dL and 11.9 g/dL in non-pregnant women.
- Moderate Anemia: Hemoglobin levels between 8 g/dL and 10.9 g/dL.
- Severe Anemia: Hemoglobin levels below 8 g/dL [135].

In addition to hemoglobin levels, clinical symptoms such as fatigue, pallor, dizziness, and shortness of breath are commonly used to assess IDA severity. Severe anemia may necessitate immediate medical interventions, including intravenous iron therapy or blood transfusion [136].

Importance of Iron in Hemoglobin Production and Oxygen Transport

Iron plays a fundamental role in erythropoiesis, the process by which red blood cells (RBCs) are produced in the bone marrow. It is a critical component of hemoglobin, the protein in RBCs responsible for oxygen transport. Hemoglobin consists of four globin chains, each containing a heme group with an iron atom at its center. This iron atom binds reversibly to oxygen in the lungs and releases it to tissues where oxygen is needed. Without sufficient iron, the body cannot produce adequate amounts of hemoglobin, leading to reduced oxygen transport capacity and subsequent anemia[79].

The process of erythropoiesis is regulated by erythropoietin, a hormone produced by the kidneys in response to hypoxia (low oxygen levels). When iron levels are low, erythropoiesis is impaired, resulting in microcytic (small) and hypochromic (pale) red blood cells. These abnormal RBCs have reduced oxygen-carrying capacity, which manifests as fatigue, pallor, and reduced physical and cognitive performance. Studies have shown that maintaining adequate iron levels is essential not only for hemoglobin synthesis but also for optimal immune function and cellular metabolism[80].

Iron Homeostasis

The human body tightly regulates iron absorption, transport, and storage to maintain a delicate balance necessary for hemoglobin synthesis and to prevent iron overload, which can be toxic. Dietary iron exists in two forms: heme iron, found in animal-based foods, and non-heme iron, found in plant-based foods. Heme iron is more readily absorbed by the body than non-heme iron. Once absorbed, iron is transported in the blood by a protein called transferrin and delivered to the bone marrow for hemoglobin production or to other tissues for storage [81].

Iron homeostasis is primarily regulated by hepcidin, a hormone produced by the liver. Hepcidin controls iron absorption by binding to ferroportin, a protein responsible for iron export from enterocytes (intestinal cells) and macrophages (cells that recycle old RBCs). When hepcidin levels are high, iron absorption from the diet decreases, and iron release from storage sites is inhibited. Conversely, when iron levels are low, hepcidin production is downregulated, allowing more iron to be absorbed and mobilized for erythropoiesis[82].

Iron is stored in the body primarily as ferritin, a protein that serves as a reservoir for readily available iron. In cases of iron deficiency, ferritin levels decrease, signaling a depletion of iron stores. Maintaining proper iron homeostasis is crucial because both iron deficiency and iron overload can lead to serious health complications. Iron deficiency results in anemia, while iron overload can cause organ damage due to oxidative stress [83].

Mechanism of Iron Absorption and Utilization

Iron plays a pivotal role in various physiological processes, including oxygen transport, energy metabolism, and DNA synthesis. The absorption of dietary iron primarily occurs in the duodenum and the proximal jejunum. Iron exists in two dietary forms: heme iron, which is predominantly found in animal-based foods and absorbed more efficiently, and non-heme iron, which is present in plant-based foods and has lower bioavailability due to dietary inhibitors such as phytates and polyphenols [96]. Heme iron is absorbed with an efficiency of 15%–35%, whereas non-heme iron has an absorption rate of 2%–20%, depending on the presence of enhancers like vitamin C [97].

Once absorbed, iron is transported across the enterocyte membrane by ferroportin, the only known cellular iron exporter. In the bloodstream, iron binds to transferrin, a plasma protein that delivers it to tissues such as the bone marrow, where it is utilized for hemoglobin synthesis. Excess iron is stored in the liver, spleen, and bone marrow as ferritin or hemosiderin, serving as a reserve during periods of increased demand or insufficient dietary intake [98].

Hepcidin, a liver-derived hormone, plays a key role in regulating iron absorption and distribution. Elevated hepcidin levels inhibit ferroportin, reducing iron absorption and mobilization from stores. Conversely, low hepcidin levels during iron deficiency or increased erythropoietic activity enhance iron absorption [99].

Stages of Iron Deficiency

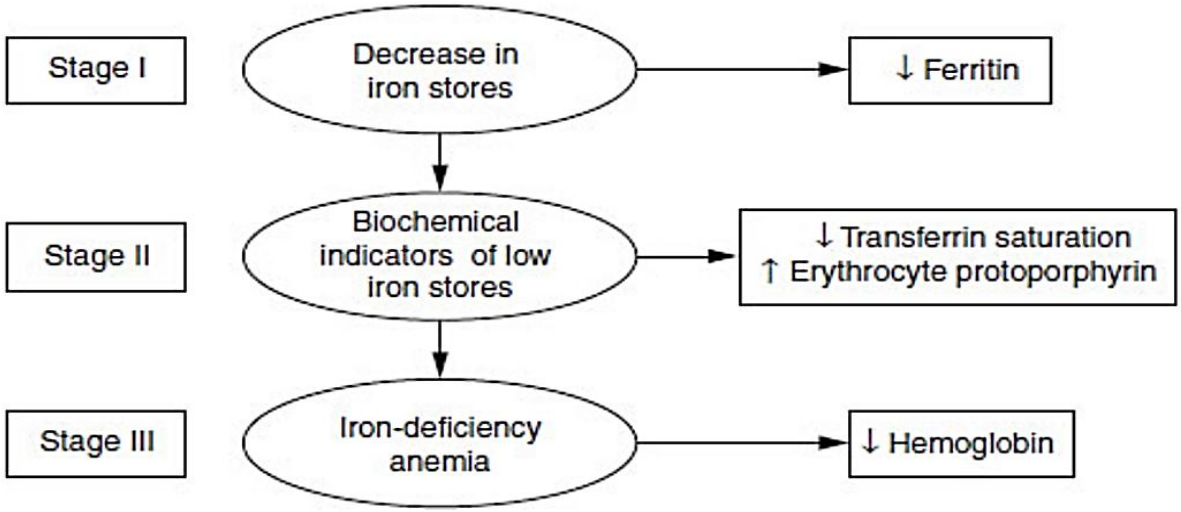


Figure 7: Stages of Iron Deficiency

a) Depletion of Iron Stores

The first stage involves the depletion of iron reserves in the liver, spleen, and bone marrow. Serum ferritin levels decrease, though hemoglobin levels remain normal. Despite the absence of overt symptoms, the body's ability to meet increased iron demands is diminished [100].

b) Iron-Deficient Erythropoiesis

During this stage, insufficient iron availability limits hemoglobin synthesis, resulting in the production of microcytic and hypochromic red blood cells. Laboratory findings include decreased serum iron levels, increased total iron-binding capacity (TIBC), and low transferrin saturation. However, clinical anemia may not yet be evident [101].

c) Iron Deficiency Anemia (IDA)

The final stage is marked by overt anemia, with hemoglobin levels falling below the diagnostic threshold (<12 g/dL in non-pregnant women and <11 g/dL in pregnant women, as defined by WHO). Clinical symptoms, including fatigue, pallor, weakness, shortness of breath, and impaired cognitive function, become evident. Diagnostic tests reveal low hemoglobin and hematocrit levels, reduced mean corpuscular volume (MCV), and low mean corpuscular hemoglobin concentration (MCHC), confirming microcytic, hypochromic anemia [102].

Laboratory Diagnosis

Diagnosis of IDA involves a combination of clinical evaluation and laboratory tests. The key parameters include:

a) Hemoglobin Levels

Low hemoglobin levels suggest anemia, but further testing is necessary to confirm iron deficiency as the underlying cause [26].

b) Serum Ferritin

Serum ferritin is a reliable indicator of iron stores in the body. A ferritin level below 15 µg/L typically indicates iron deficiency in non-pregnant women. However, ferritin may be elevated during inflammatory conditions, which can reduce its diagnostic accuracy [137].

c) Transferrin Saturation

Transferrin saturation measures the percentage of transferrin that is bound to iron. A value below 20% is suggestive of iron deficiency [138].

d) Total Iron-Binding Capacity (TIBC)

Elevated TIBC is another indicator of IDA, reflecting the body's increased effort to bind and transport available iron [136].

e) Peripheral Blood Smear

A peripheral blood smear typically reveals microcytic (small) and hypochromic (pale) red blood cells, which are characteristic of IDA [139].

f) RBC Indices

Definition of RBC Indices

RBC indices are a set of key parameters used to evaluate the size, hemoglobin content, and concentration of red blood cells (RBCs). These indices are crucial in diagnosing and classifying various types of anemia, such as iron deficiency anemia (IDA) and thalassemia [140].

Mean Corpuscular Volume (MCV)

MCV measures the average size of red blood cells and is expressed in femtoliters (fL). It helps differentiate between different types of anemia:

- Microcytic anemia: Low MCV (<80 fL), typically seen in IDA [141].
- Normocytic anemia: Normal MCV (80–100 fL).
- Macrocytic anemia: High MCV (>100 fL), observed in megaloblastic anemia due to vitamin B12 or folate deficiency.

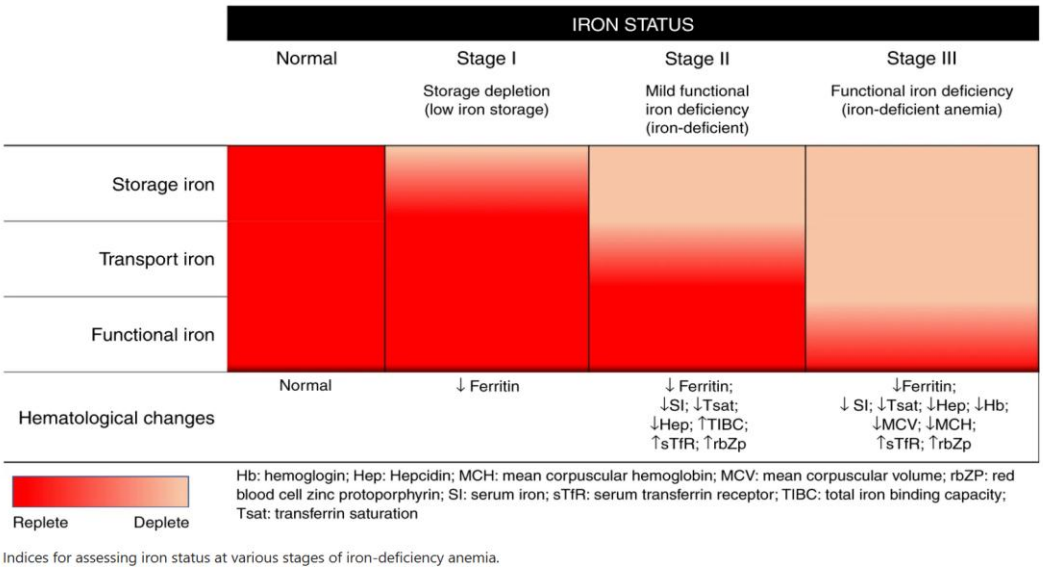
Mean Corpuscular Hemoglobin (MCH)

MCH represents the average amount of hemoglobin per RBC, expressed in picograms (pg). Low MCH (<27 pg) suggests hypochromia, a hallmark of IDA. Normal or elevated MCH may indicate normochromic or macrocytic anemia [142].

Mean Corpuscular Hemoglobin Concentration (MCHC)

MCHC measures the average concentration of hemoglobin in a given volume of RBCs and is expressed in g/dL.

- Low MCHC (<32 g/dL): Indicates hypochromic anemia, common in IDA.
- Normal MCHC (32–36 g/dL): Seen in normochromic anemia.
- High MCHC, though rare, may be seen in hereditary spherocytosis [143].


IRON STATUS
Normal
Stage I
Storage depletion (low iron storage)
Stage II
Mild functional iron deficiency (iron-deficient)
Stage III
Functional iron deficiency (iron-deficient anemia)
Storage iron
Transport iron
Functional iron
Hematological changes
Normal
↓ Ferritin
↓ Ferritin; ↓SI; ↓Tsat; ↓Hep; ↑TIBC; ↑sTfR; ↑rbZp
↓Ferritin; ↓ SI; ↓Tsat; ↓Hep; ↓Hb; ↓MCV; ↓MCH; ↑sTfR; ↑rbZp
Replete
Deplete
Hb: hemoglogin; Hep: Hepcidin; MCH: mean corpuscular hemoglobin; MCV: mean corpuscular volume; rbZP: red blood cell zinc protoporphyrin; SI: serum iron; sTfR: serum transferrin receptor; TIBC: total iron binding capacity; Tsat: transferrin saturation

Indices for assessing iron status at various stages of iron-deficiency anemia.

Figure 8: ***RBC Indices in Iron Deficiency Anemia (IDA)***

RBC Indices in Iron Deficiency Anemia (IDA)

In IDA, RBC indices display characteristic changes that aid in diagnosis:

- Low MCV: IDA presents as microcytic anemia with MCV typically <80 fL due to impaired hemoglobin synthesis from insufficient iron [141].
- Low MCH: The decreased hemoglobin content results in pale (hypochromic) RBCs, with MCH <27 pg [140].
- Low MCHC: IDA also leads to a reduced hemoglobin concentration, with MCHC typically <32 g/dL, resulting in hypochromia that can be observed in a peripheral blood smear [144]. These indices are instrumental in differentiating IDA from other forms of anemia, such as anemia of chronic disease (normocytic, normochromic) and megaloblastic anemia (macrocytic, normochromic). A combination of low MCV, MCH, and MCHC is highly indicative of IDA and prompts further investigation through serum ferritin, transferrin saturation, and total iron-binding capacity (TIBC) to confirm the diagnosis [142].

Hepcidin as a Non-Invasive Marker

Hepcidin is a peptide hormone primarily synthesized by hepatocytes in the liver, playing a central role in iron metabolism and homeostasis. By regulating iron absorption in the intestines and its release from macrophages and hepatocytes, hepcidin maintains iron balance within the body. Its production is finely tuned by iron levels, erythropoietic activity, and inflammatory stimuli, making it a dynamic marker of iron-related disorders [145]. Given its significant role in the regulation of iron, hepcidin has emerged as a non-invasive biomarker for assessing various iron states, particularly iron deficiency anemia (IDA) and anemia of chronic disease (ACD). Its utility lies in its ability to reflect the underlying pathophysiology of iron metabolism in these conditions, enabling differential diagnosis and better treatment strategies. Hepcidin is a small peptide hormone consisting of 25 amino acids, primarily synthesized by the liver. It is encoded by the HAMP gene and initially produced as a preprohormone, undergoing post-translational modifications to form the biologically active hormone [153]. Hepcidin circulates in the bloodstream and acts as the key regulator of systemic iron homeostasis by interacting with ferroportin, the only known cellular iron exporter [154].

Hepcidin: Structure and Synthesis

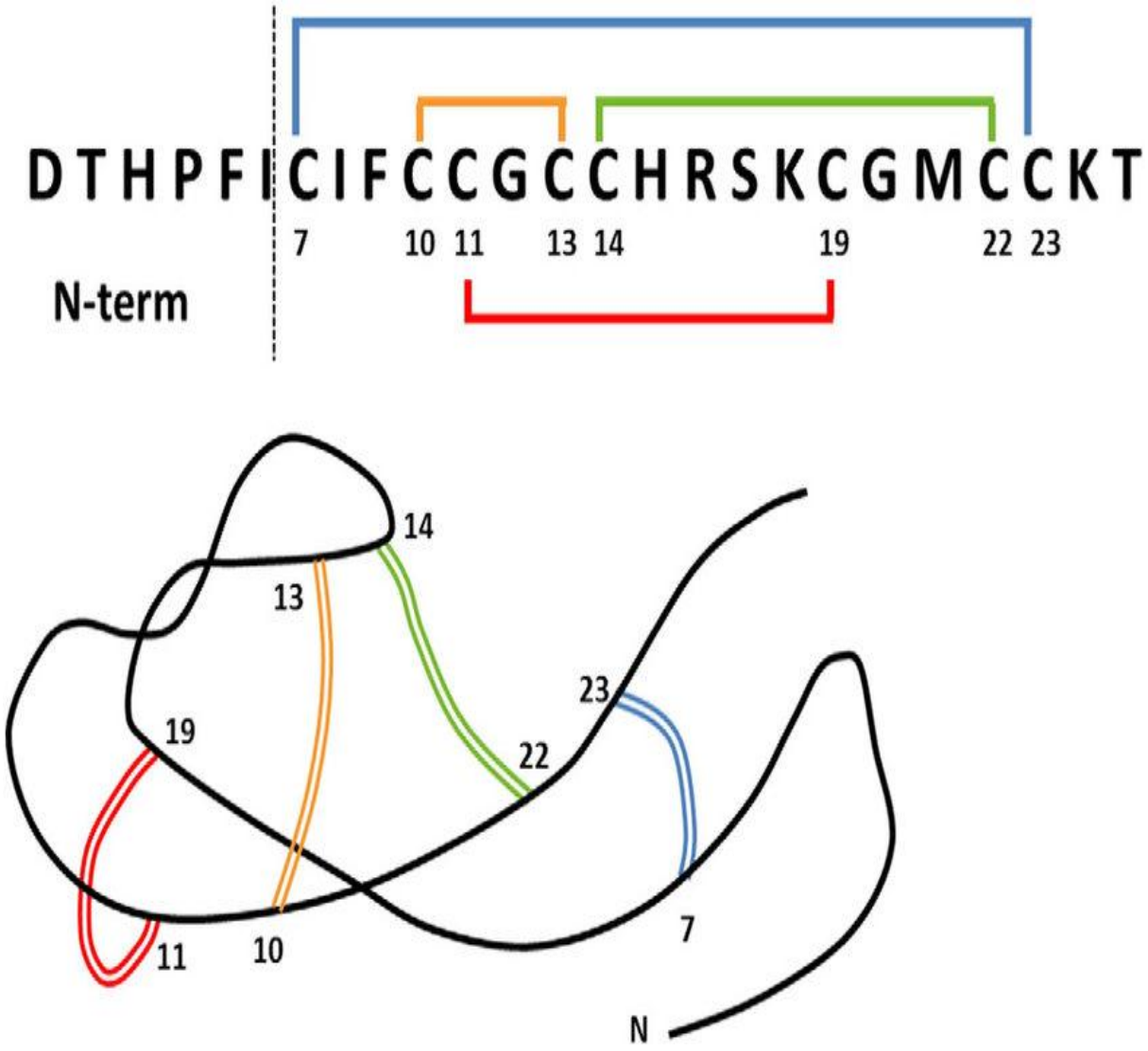


Fig.9: Hepcidin: the amino-acid sequence and structure. Hepcidin is synthesized as an 84-amino-acid (AA) preproprotein. A furin-like proprotein convertase is responsible for cleavage of pro-hepcidin to hepcidin. The human 25-amino-acid hepcidin peptide contains four disulphide bonds. N-terminal domain interacts with ferroportin.

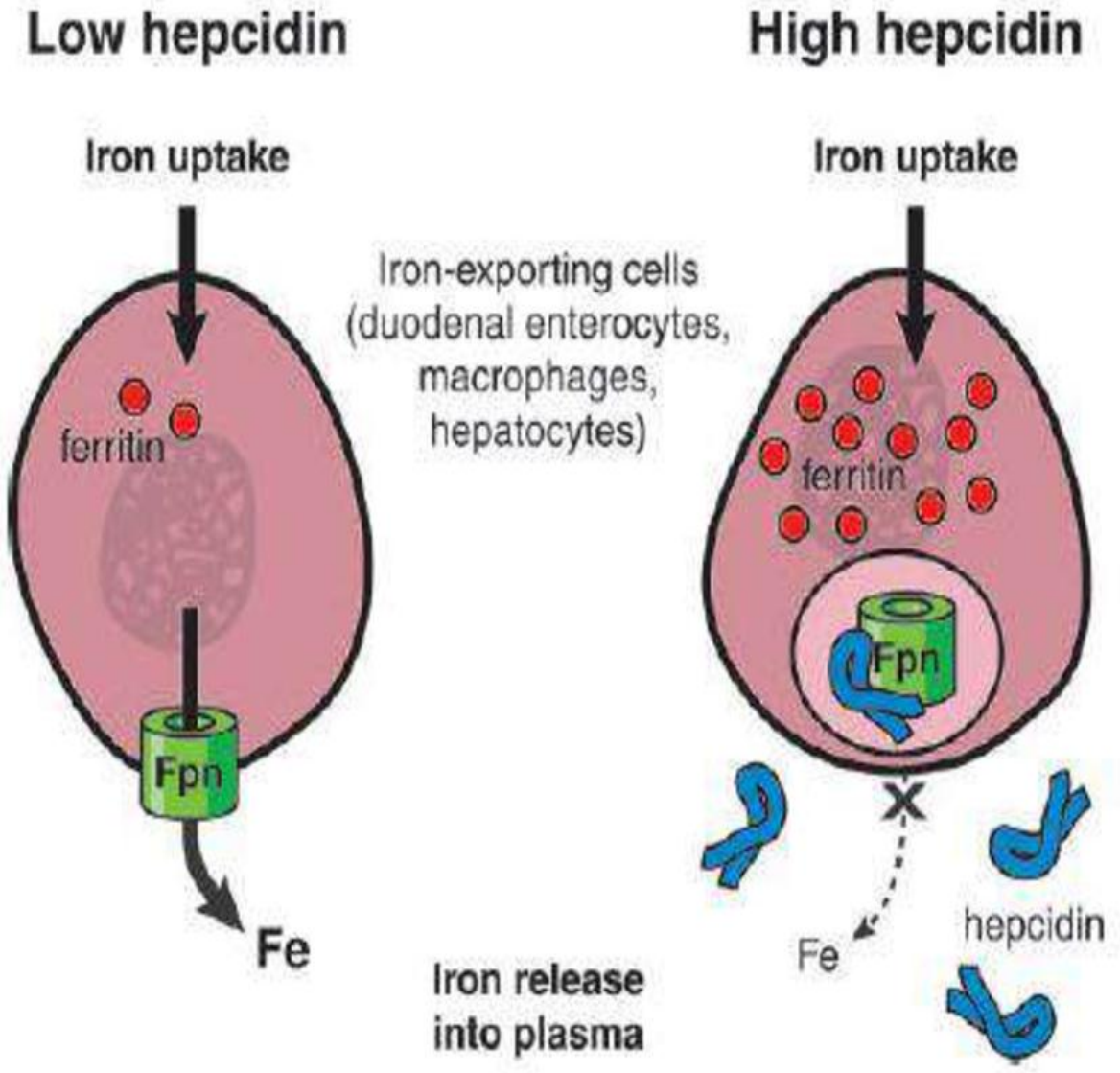


Figure 10: The mechanism of hepcidin-mediated cellular iron regulation

Regulation by Iron and Inflammation

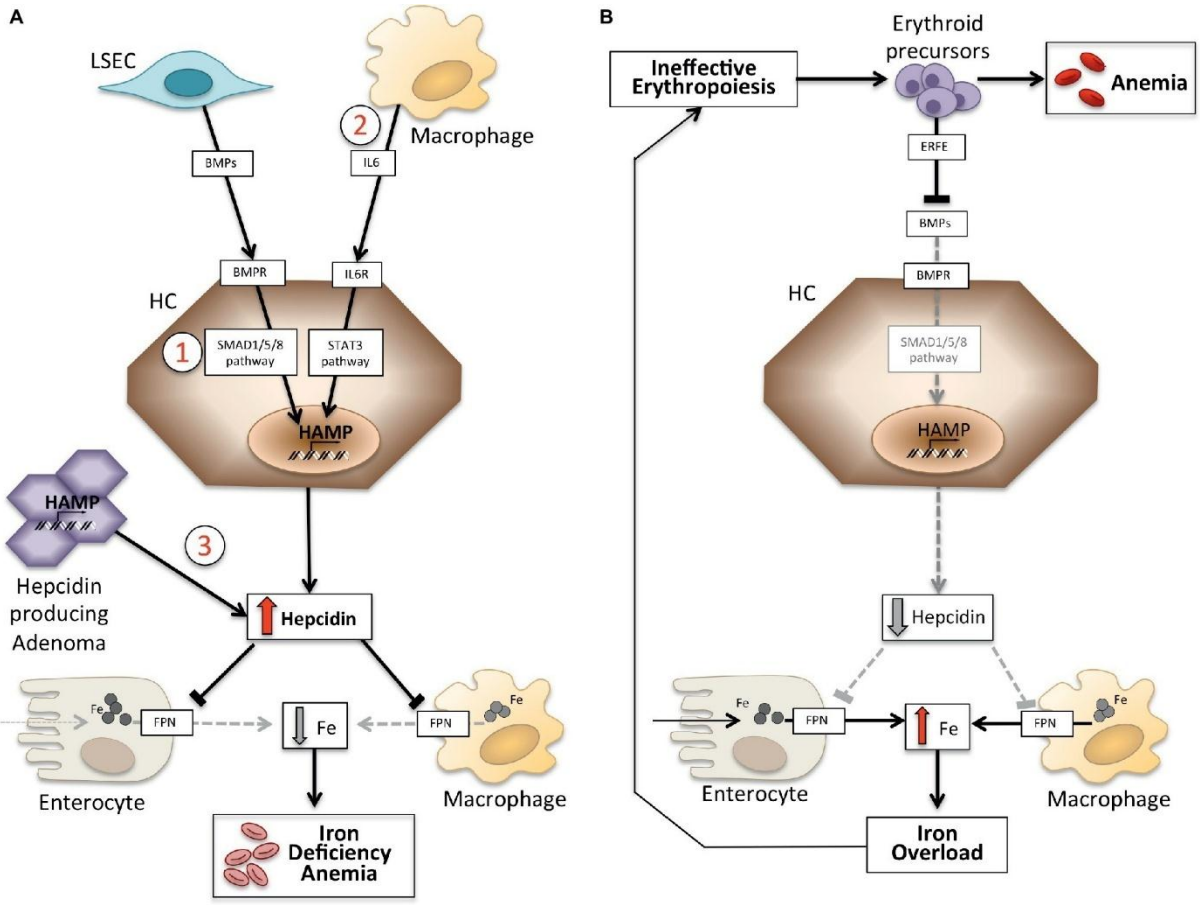


Figure 11: Schematic representation of mechanisms of anemias with high (left panel) and low hepcidin (right panel). Panel (A). Molecular pathogenesis of anemia associated with high hepcidin levels. LSEC, liver sinusoidal endothelial cells producing bone morphogenetic proteins (BMPs); BMPRs, BMP receptors; IL6, interleukin 6; HC, hepatocytes; HAMP, hepcidin gene. Fe, iron; FPN, ferroportin; 1, IRIDA; 2, Anemia of inflammation; 3, hepcidin producing adenoma. Panel (B). Molecular pathogenesis of hepcidin variation in anemias due to ineffective erythropoiesis. ERFE, erythroferrone sequestering BMPs.

Hepcidin Levels in Different Iron States

1.In Iron-Replete Individuals

In individuals with sufficient or excessive iron stores, hepcidin production is upregulated. This increased hepcidin level inhibits iron absorption in the duodenum by binding to and inducing the internalization and degradation of ferroportin, the sole iron exporter on enterocytes and macrophages. Additionally, hepcidin limits the release of iron from macrophages and hepatocytes, thereby preventing iron overload [146]. Elevated hepcidin levels in iron-replete individuals reflect a feedback mechanism aimed at maintaining homeostasis by reducing circulating iron levels.

2. In Iron Deficiency

During states of iron deficiency, the body's demand for iron increases to support essential physiological processes such as erythropoiesis. In response, hepcidin synthesis is downregulated, promoting increased dietary iron absorption and mobilization of stored iron from macrophages and hepatocytes [147]. Low circulating hepcidin levels serve as an adaptive response to restore iron availability. This reduction in hepcidin is characteristic of iron deficiency anemia (IDA), where the lack of iron is primarily due to insufficient dietary intake, malabsorption, or chronic blood loss. Measuring hepcidin levels can provide a reliable biomarker for diagnosing IDA, as low hepcidin is directly correlated with depleted iron stores.

3. In Anemia of Chronic Disease (ACD)

Anemia of chronic disease (ACD), also referred to as anemia of inflammation, is characterized by impaired iron utilization due to sustained inflammatory responses. In this condition, hepcidin synthesis is upregulated, primarily driven by inflammatory cytokines such as interleukin-6 (IL-6). Elevated hepcidin levels in ACD contribute to iron sequestration within macrophages and reduced iron availability for erythropoiesis, resulting in anemia [148]. The chronic activation of hepcidin in ACD reflects the body's attempt to restrict iron availability to pathogens, as part of the immune response during inflammation.Elevated hepcidin levels in ACD differentiate it from IDA, where hepcidin levels are typically low. This distinction is clinically important, as the management of these conditions differs

significantly. For example, iron supplementation is beneficial in IDA but may exacerbate the inflammatory response in ACD [149].

4. Iron Overload

In conditions of iron overload, hepcidin expression is upregulated. This increased production of hepcidin leads to the internalization and degradation of ferroportin, thereby reducing dietary iron absorption and iron release from macrophages and hepatocytes. This regulation prevents excess iron accumulation, protecting the body from iron toxicity [11].

5. Inflammation

Inflammatory cytokines, particularly interleukin-6 (IL-6), stimulate hepcidin production during infection or chronic inflammation. Elevated hepcidin levels during inflammation restrict iron availability, contributing to anemia of chronic disease (ACD) despite adequate iron stores. This response is thought to limit iron availability to pathogens, although it also impairs erythropoiesis [156].

6. Role in Iron Homeostasis

Hepcidin is crucial in maintaining iron balance by regulating ferroportin activity, thereby controlling iron efflux from enterocytes, hepatocytes, and macrophages [157].

7. Binding to Ferroportin

Hepcidin binds to ferroportin, the transmembrane protein responsible for exporting iron from cells. Upon binding, hepcidin induces the internalization and lysosomal degradation of ferroportin, thus blocking iron export into the bloodstream [158].

Promoting Ferroportin Degradation

This degradation mechanism decreases dietary iron absorption and inhibits iron release from macrophages that recycle senescent red blood cells, leading to reduced serum iron levels [159]. Disruptions in hepcidin expression or function can cause iron metabolism disorders, such as iron-refractory iron deficiency anemia (IRIDA), where hepcidin levels remain inappropriately high, or hereditary hemochromatosis, characterized by low hepcidin levels and excessive iron accumulation [160].

Why Urinary Hepcidin?

Reason for Choosing Urinary Hepcidin over Serum Hepcidin

1. **Non-Invasive Nature**: Urinary hepcidin collection is non-invasive, painless, and more convenient compared to venipuncture required for serum hepcidin. This non-invasive approach is particularly useful in large-scale epidemiological studies, pediatric populations, and resource-limited settings where blood collection may be challenging [86].

2. **Ease of Collection:** Urine samples can be collected easily and repeatedly without the need for trained healthcare professionals, unlike serum samples. This makes urinary hepcidin suitable for frequent monitoring of iron status, especially in vulnerable populations such as children and pregnant women [85].

3. **Cost-Effectiveness**: The logistics of collecting, storing, and transporting urine samples are more cost-effective than those of serum samples. This advantage reduces the overall cost of large-scale anemia screening and management programs [161].

4. **Correlation with Iron Status**: Several studies have demonstrated a significant correlation between urinary hepcidin levels and iron status. Low urinary hepcidin levels indicate iron deficiency, whereas elevated levels suggest adequate or excessive iron stores. Urinary hepcidin has shown promising diagnostic accuracy for early detection of iron deficiency anemia (IDA) [162] [86].

5. **Suitability for Public Health Programs**: Due to its ease of collection and lower costs, urinary hepcidin is ideal for integration into public health programs aimed at early detection and management of IDA in high-burden areas. Its application in population-level screening could improve anemia control efforts in resource-limited settings [87].

Urinary hepcidin presents a practical, non-invasive, and reliable biomarker for assessing iron status and diagnosing iron deficiency anemia. Its advantages in terms of ease of collection, cost-effectiveness, and potential use in large-scale public health programs make it a promising tool for improving anemia management. Future research focusing on standardizing urinary hepcidin assays and establishing reference ranges will enhance its clinical utility.

METHODOLOGY

Study Design and Setting

This study was conducted as an observational study to evaluate the role of urinary hepcidin as a non-invasive biomarker for iron deficiency anemia (IDA). The study was carried out at the Department of Biochemistry and Pathology, Santosh Medical College, Ghaziabad, over a duration of one year. The study was approved by the Institutional Ethics Committee (IEC), and all ethical guidelines were strictly followed.

Sample Size Calculation

The sample size was determined based on the estimated prevalence of iron deficiency anemia (IDA) in young adult females in North India, which was reported as 29.9%. The following formula was used for sample size calculation:

$$n = \frac{DEFF \times Np(1-p)}{(d^2/Z^2_{1-\alpha/2} \times (N-1) + p(1-p))}$$

where:

- **N** = Population size (1,000,000)
- **p** = Hypothesized prevalence of IDA (**29.9% ± 10**)
- **d** = Confidence limit (**10%**)
- **Design effect (DEFF)** = 1

The calculated sample sizes at different confidence levels were:

- **95% confidence level** = **81 samples**
- **99% confidence level** = **140 samples**
- **99.9% confidence level** = **227 samples** (Final chosen sample size)

A total of 227 samples were collected to ensure statistical power and to account for possible dropouts or exclusions.

Inclusion and Exclusion Criteria

Inclusion Criteria

Participants who met the following criteria were included in the study:

1. Females aged between 18-25 years.
2. Hemoglobin (Hb) levels <12 g/dL, indicative of anemia.
3. Serum ferritin levels ≤ 15 μg/L, confirming iron deficiency.
4. C-reactive protein (CRP) levels <5 mg/L, ensuring no active infection or inflammation.
5. Non-pregnant females, to eliminate physiological variations in iron metabolism.

Exclusion Criteria

The following individuals were excluded from the study:

1. Females receiving iron therapy, as supplementation could alter hepcidin levels.
2. Females using oral contraceptives, which might influence iron metabolism.
3. Individuals diagnosed with chronic diseases such as liver disease, kidney disease, or any systemic illness affecting iron homeostasis.
4. Females with malignancies, to avoid confounding factors related to altered iron metabolism in cancer.
5. Non-anemic females, as the study aimed to evaluate urinary hepcidin in IDA cases.

Ethical Considerations and Consent Process

Prior to sample collection, ethical clearance was obtained from the Institutional Ethics Committee (IEC) of Santosh Medical College. All participants were given detailed information regarding the study, including its purpose, methodology, risks, and benefits. Written informed consent was obtained from each participant before enrollment.

Participants were assured of confidentiality, and all data were handled in compliance with Good Clinical Practice (GCP) guidelines. They were also informed of their right to withdraw from the study at any stage without consequences.

Sample Collection and Storage

Blood Sample Collection

- Venous blood samples (5 mL) were collected from each participant under aseptic conditions using vacutainer tubes.
- Serum separation was performed by centrifuging the blood samples at 3000 rpm for 10 minutes at 4°C.
- Serum was stored at -80°C until further biochemical analysis.

Urine Sample Collection

- Midstream morning urine samples were collected in sterile urine containers to minimize diurnal variations in hepcidin levels.
- Urine samples were stored at -80°C after centrifugation at 3000 rpm for 10 minutes to remove debris and contaminants.

Biochemical Parameters and Analytical Methods

The following hematological and biochemical markers were analyzed to assess iron deficiency anemia and urinary hepcidin levels:

Parameter	**Method Used**	**Equipment Used**
Complete Blood Count (CBC)	**Impedance Method (Cyanide-Free Reagent for Hemoglobin Estimation)**	Automated Hematology Analyzer
Serum Iron	**Ferrozine Method**	Colorimetric Analyzer
Total Iron Binding Capacity (TIBC)	**Calculated as Serum Iron × 100 / TIBC**	Automated Chemistry Analyzer
Serum Ferritin	**Sandwich Electrochemiluminescence Assay (ECLIA)**	Immunoassay Analyzer
Transferrin Saturation	**Calculated from Serum Iron & TIBC**	Calculated Parameter
Urinary Hepcidin	**Sandwich Enzyme-Linked Immunosorbent Assay (ELISA) Method**	ELISA Plate Reader
C-Reactive Protein (CRP)	**Turbidometry**	Immunoturbidometry Analyzer

Data Management and Statistical Analysis

- **Data Entry and Cleaning:**
    - All collected data were entered into Microsoft Excel and later transferred to STATA software for analysis.
    - Missing or inconsistent data were cross-checked with the original records.
- **Descriptive Statistics:**
    - Continuous variables (e.g., serum iron, hepcidin levels, ferritin) were expressed as mean ± standard deviation (SD).
    - Categorical variables (e.g., presence or absence of anemia) were presented as frequencies and percentages.
- **Comparative Analysis:**
    - The Student's t-test was used to compare mean values of urinary hepcidin between IDA and control groups.
    - Pearson’s correlation coefficient was used to assess the relationship between urinary hepcidin and serum iron markers.
    - The receiver operating characteristic (ROC) curve was plotted to determine the diagnostic accuracy of urinary hepcidin for detecting IDA.
- **Significance Level:**
    - A p-value <0.05 was considered statistically significant.

Quality Control Measures

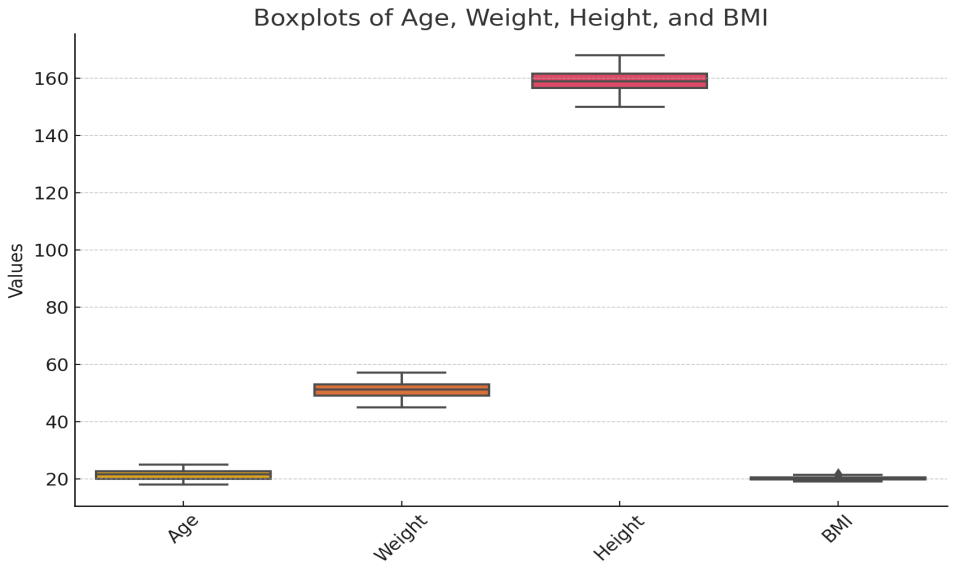
To ensure reliability and accuracy of results:

1. All laboratory tests were performed in duplicate, and mean values were recorded.
2. Standardized protocols and calibration of equipment were maintained throughout the study.
3. Internal and external quality control samples were run with every batch of tests.
4. Blinded assessment was performed for sample processing to minimize bias.

RESULTS

Table 1: Demographic and Clinical Characteristics of Study Participants

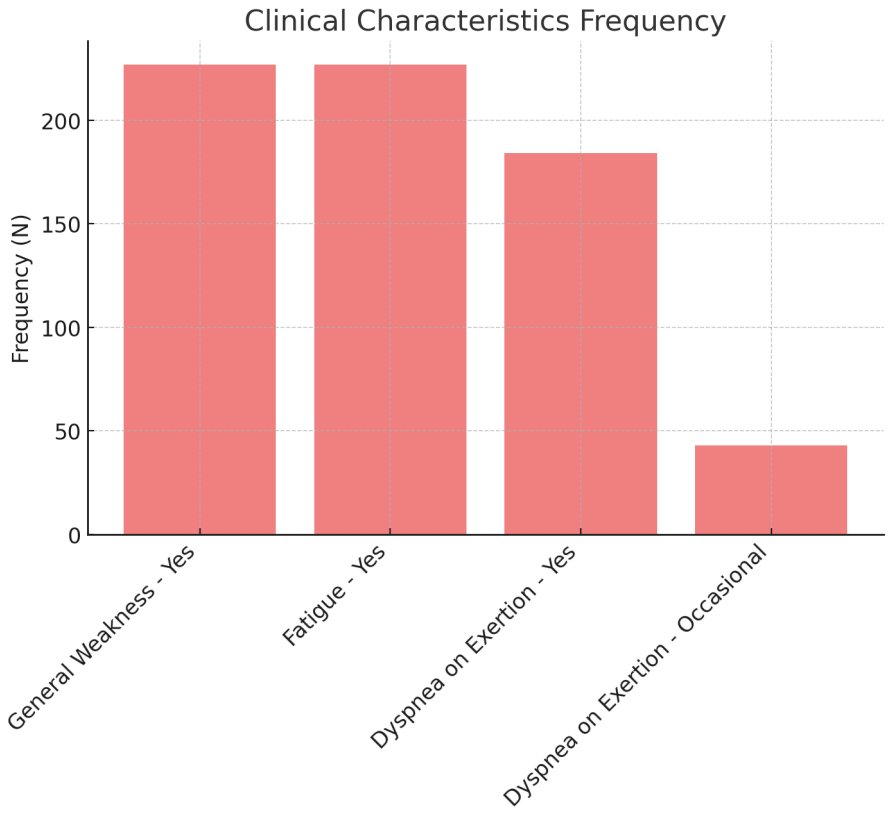
Parameter	**Mean ± SD**	**Range (Min-Max)**
Age (years)	21.40 ± 2.12	18 - 25
Weight (kg)	51.06 ± 2.89	45 - 57
Height (cm)	159.30 ± 3.62	150 - 168
BMI (kg/m²)	20.12 ± 0.51	18.90 - 22.00
Socioeconomic Status	N (%)	
Low	119 (52.4%)	
Middle	108 (47.6%)	

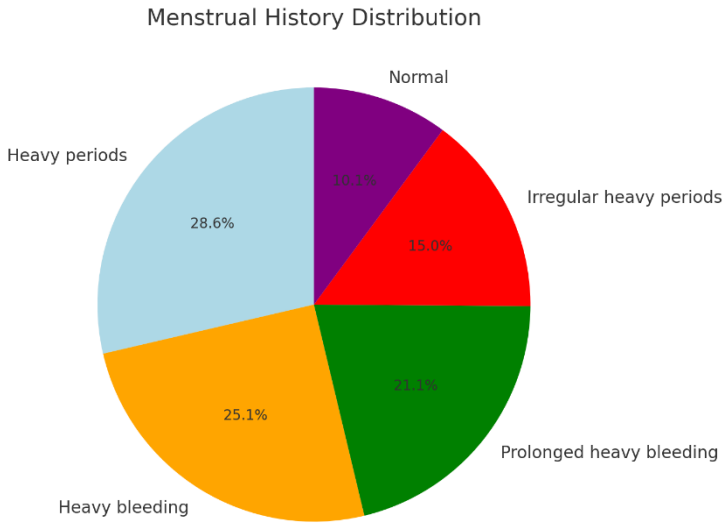


The study cohort comprises young reproductive-age females (mean age: 21.40 ± 2.12 years), with a lean body profile (BMI: 20.12 ± 0.51 kg/m²), making them physiologically vulnerable to iron deficiency anemia (IDA). A significant proportion (52.4%) belongs to the low socioeconomic status (SES) category, highlighting potential nutritional insufficiencies as a key contributing factor to IDA. The average weight (51.06 ± 2.89 kg) and height (159.30 ± 3.62 cm) suggest an overall healthy but possibly undernourished group. Given the narrow BMI range (18.90 - 22.00 kg/m²), subclinical nutritional deficiencies could be prevalent, compounding anemia risk. These findings emphasize the need for targeted interventions focusing on nutritional education, supplementation, and socioeconomic support to mitigate IDA prevalence in this demographic.

Table 2: Clinical Characteristics Frequency

Clinical Characteristic	Frequency (N)	Percentage (%)
General Weakness – Yes	227	100.0
Fatigue – Yes	227	100.0
Dyspnea on Exertion – Yes	184	81.1
Dyspnea on Exertion – Occasional	43	18.9
Menstrual History		
Heavy periods	65	28.6
Heavy bleeding	57	25.1
Prolonged heavy bleeding	48	21.1
Irregular heavy periods	34	15.0
Normal	23	10.1

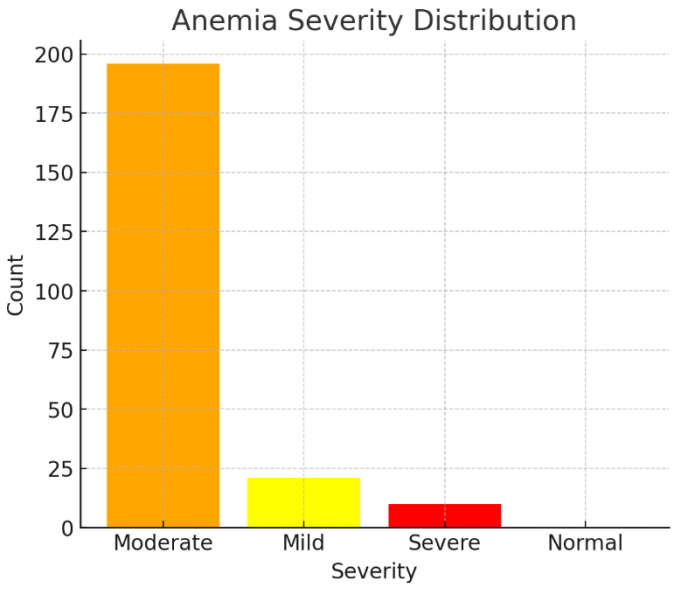




The universal presence of fatigue and general weakness (100%) among participants strongly suggests severe symptomatic iron deficiency anemia (IDA), a common issue in reproductive-age females. Dyspnea on exertion (81.1%), with an additional 18.9% experiencing occasional episodes, indicates a significant reduction in oxygen-carrying capacity due to low hemoglobin levels. Menstrual blood loss emerges as a major contributing factor, with 28.6% reporting heavy periods, 25.1% heavy bleeding, 21.1% prolonged heavy bleeding, and 15.0% irregular heavy periods, highlighting the role of excessive menstrual flow in exacerbating anemia. Only 10.1% of participants reported normal menstrual patterns, reinforcing the link between abnormal menstruation and IDA prevalence. These findings emphasize the need for targeted screening, menstrual regulation strategies, and iron supplementation programs to address IDA in this demographic.

Table 3: Anemia Severity Distribution

Anemia Severity	Count	Percentage (%)
Moderate	196	86.3
Mild	21	9.3
Severe	10	4.4



The distribution of anemia severity among participants reveals a high prevalence of moderate anemia (86.3%), followed by mild anemia (9.3%), and severe anemia in a smaller subset (4.4%).

Moderate anemia (196 participants, 86.3%) is the most common, indicating widespread but non-life-threatening iron deficiency, which still requires intervention to prevent progression to severe anemia.

Mild anemia (21 participants, 9.3%) suggests early-stage iron deficiency, which may be more manageable through dietary modifications and supplementation.

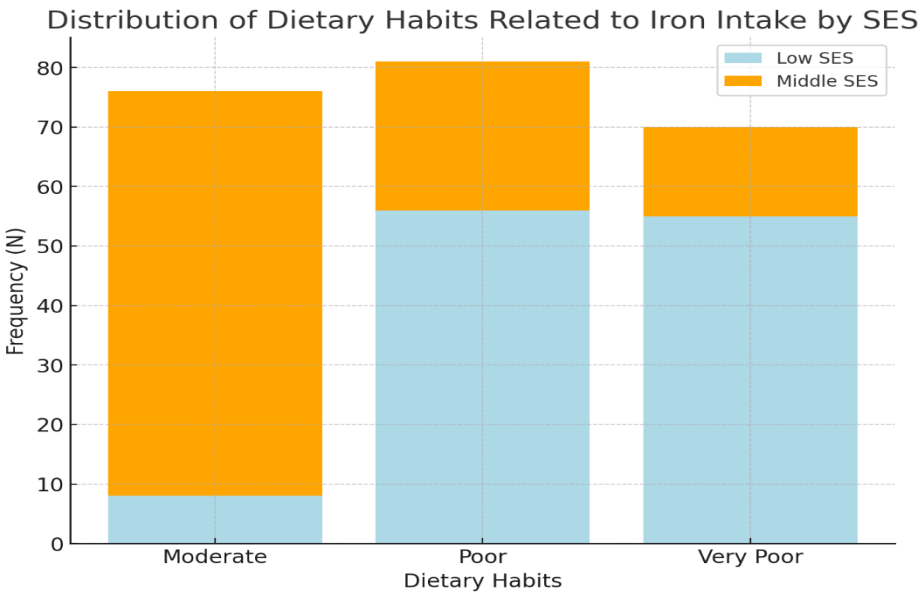
Severe anemia (10 participants, 4.4%) represents the most critical cases, likely requiring urgent medical intervention, intravenous iron therapy, or blood transfusions to restore hemoglobin levels and prevent complications.

Table 4: Distribution of Dietary Habits Related to Iron Intake

Dietary Habits (Iron Intake)	Low SES N (%)	Middle SES N (%)	Total N (%)
Moderate	8 (6.7%)	68 (63.0%)	76 (33.5%)
Poor	56 (47.1%)	25 (23.2%)	81 (35.7%)
Very Poor	55 (46.2%)	15 (13.9%)	70 (30.8%)
Total	119 (100.0%)	108 (100.0%)	227 (100.0%)

Chi-square Test Results

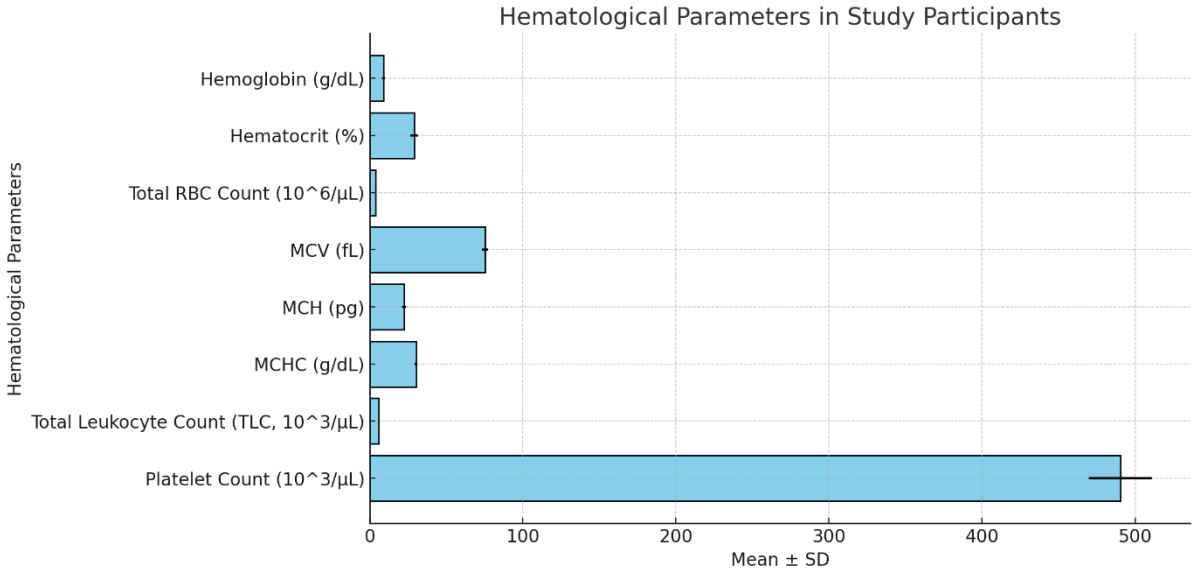
- p-value: 0.0000



The majority of low SES participants (93.3%) exhibit poor or very poor iron intake, compared to a higher prevalence of moderate intake (63.0%) in the middle SES group, emphasizing the strong link between socioeconomic status and dietary quality. Overall, 66.5% of participants have poor or very poor iron intake, indicating that inadequate dietary iron is a primary contributor to iron deficiency anemia (IDA) in this cohort. The highly significant chi-square result (p=0.0000) confirms that dietary disparities based on SES play a crucial role in anemia severity. The greater burden of poor nutrition among low SES individuals highlights the need for iron-rich dietary interventions, fortified food programs, and economic support to improve anemia outcomes. Addressing these nutritional gaps through education and supplementation strategies could be critical in reducing IDA prevalence in young reproductive-age females.

Table 5: Hematological Parameters in Study Participants

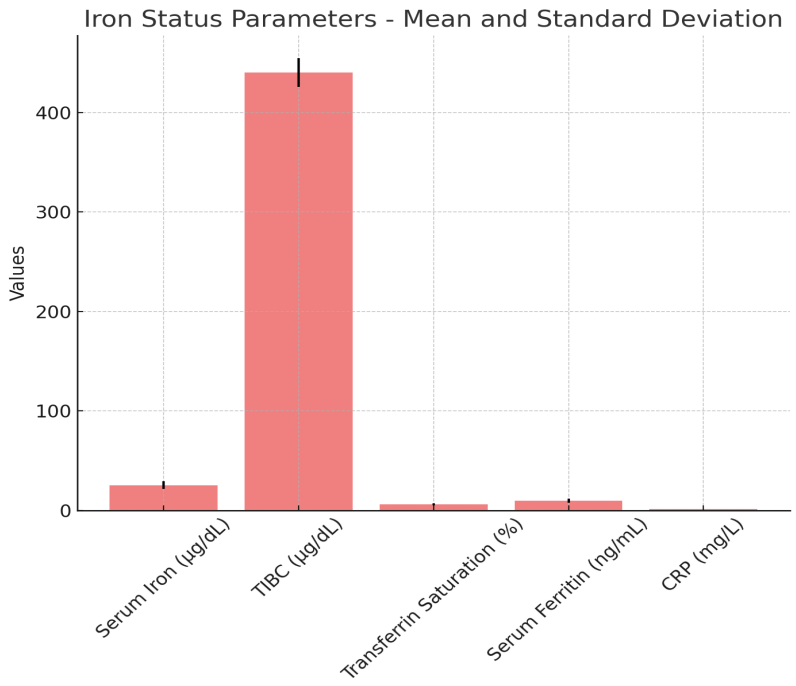
Parameter	Mean ± SD	Range (Min-Max)	95% CI
Hemoglobin (g/dL)	8.94 ± 0.94	6.10 - 11.50	(8.82, 9.06)
Hematocrit (%)	29.02 ± 2.51	18.00 - 34.50	(28.66, 29.38)
Total RBC Count (10^6/µL)	3.52 ± 0.26	2.50 - 4.10	(3.49, 3.55)
MCV (fL)	75.16 ± 1.95	68.00 - 80.00	(74.90, 75.41)
MCH (pg)	22.37 ± 1.43	16.00 - 26.00	(22.19, 22.56)
MCHC (g/dL)	30.22 ± 0.96	27.00 - 33.00	(30.10, 30.34)
Total Leukocyte Count (TLC, 10^3/µL)	5.89 ± 0.22	5.20 - 6.50	(5.86, 5.92)
Platelet Count (10^3/µL)	490.31 ± 20.42	450.00 - 540.00	(487.65, 492.97)



The mean hemoglobin level (8.94 ± 0.94 g/dL) confirms the presence of moderate to severe anemia, well below the normal threshold of 12 g/dL, characteristic of iron deficiency anemia (IDA). The low MCV (75.16 ± 1.95 fL) and MCH (22.37 ± 1.43 pg) indicate a microcytic and hypochromic profile, a hallmark of IDA due to insufficient hemoglobin synthesis. Elevated platelet counts (490.31 ± 20.42 × 10^3/µL) suggest compensatory thrombocytosis, likely triggered by chronic blood loss from heavy menstrual bleeding, reinforcing its role in anemia progression. The narrow confidence intervals (e.g., Hb 8.82-9.06 g/dL) suggest a consistently anemic profile across the study cohort, with little variation. These findings emphasize the critical need for targeted iron supplementation, dietary interventions, and menstrual regulation strategies to mitigate IDA's impact on reproductive-age females.

Table 6: Iron Status Parameters in Study Participants

Parameter	Mean ± SD	Range (Min-Max)	95% CI
Serum Iron (µg/dL)	25.31 ± 3.87	11.00 - 35.00	(24.81, 25.82)
TIBC (µg/dL)	440.42 ± 14.56	405.00 - 490.00	(438.52, 442.31)
Transferrin Saturation (%)	5.78 ± 1.03	2.20 - 8.60	(5.64, 5.91)
Serum Ferritin (ng/mL)	9.48 ± 1.99	3.00 - 14.00	(9.22, 9.73)
CRP (mg/L)	1.37 ± 0.20	1.00 - 2.10	(1.34, 1.39)

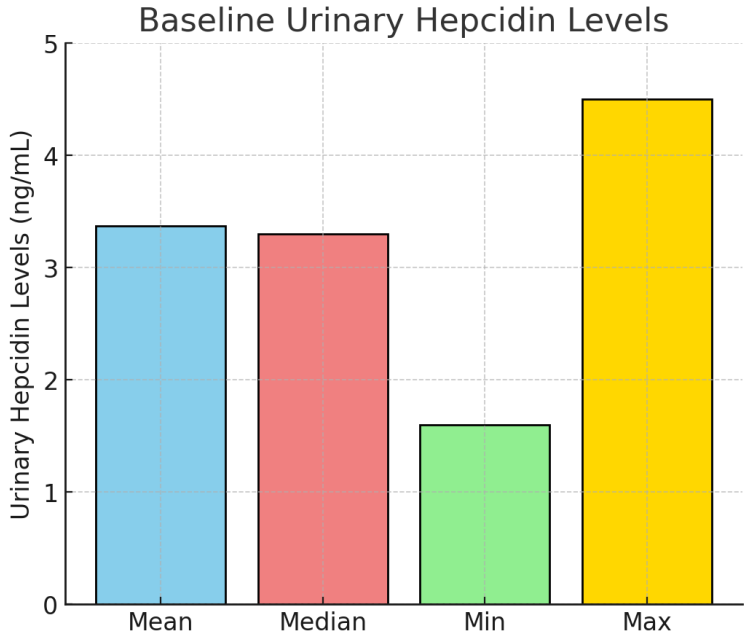


The low serum iron (25.31 ± 3.87 µg/dL), ferritin (9.48 ± 1.99 ng/mL), and transferrin saturation (5.78 ± 1.03%) confirm severe iron deficiency anemia (IDA), which is highly prevalent in young reproductive-age females. The elevated total iron-binding capacity (TIBC, 440.42 ± 14.56 µg/dL) indicates iron scarcity, a hallmark feature of menstrual-related chronic blood loss contributing to anemia. Severely depleted ferritin levels further confirm exhausted iron stores, reinforcing the need for urgent iron replenishment strategies. The mildly elevated C-reactive protein (CRP, 1.37 ± 0.20 mg/L) suggests minimal inflammation, ruling out anemia of chronic disease and supporting IDA as the primary cause of anemia in this cohort. These findings highlight the necessity of iron supplementation, dietary interventions, and menstrual regulation strategies to prevent worsening IDA and its long-term health consequences.

Table 7: Baseline Urinary Hepcidin Levels

Overall Distribution

Category	Statistic	Value
Urinary Hepcidin Levels	Mean	3.37
	Median	3.30
	Range (Min-Max)	1.60 - 4.50



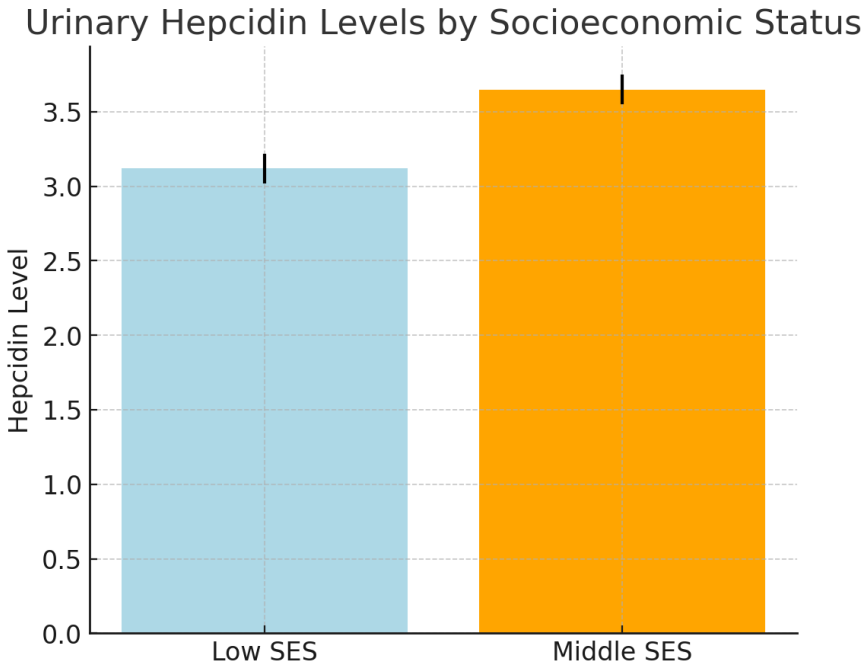
The mean urinary hepcidin level (3.37 ng/mL) is suppressed, aligning with the expected downregulation of hepcidin in iron deficiency anemia (IDA), as the body attempts to enhance iron absorption and mobilization. Participants from low SES groups exhibit significantly lower hepcidin levels (3.12 vs. 3.65 ng/mL, p=0.0000), correlating with more severe anemia due to chronic dietary iron insufficiency. Furthermore, hepcidin levels progressively decline with worsening iron intake (moderate 3.77 vs. very poor 3.05 ng/mL, p=0.0000), reinforcing the direct impact of diet on iron regulation. This hepcidin suppression pattern highlights severe functional iron deficiency, emphasizing the need for nutritional interventions, iron supplementation, and improved dietary diversity to restore iron homeostasis. Addressing these disparities through targeted public health strategies is crucial to mitigating IDA burden in young reproductive-age females.

Table 8.Distribution by Socioeconomic Status

Socioeconomic Status	Mean	Median	Range (Min-Max)
Low	3.12	3.2	1.60 - 4.20
Middle	3.65	3.6	2.90 - 4.50

T-test for SES groups (Low vs Middle):

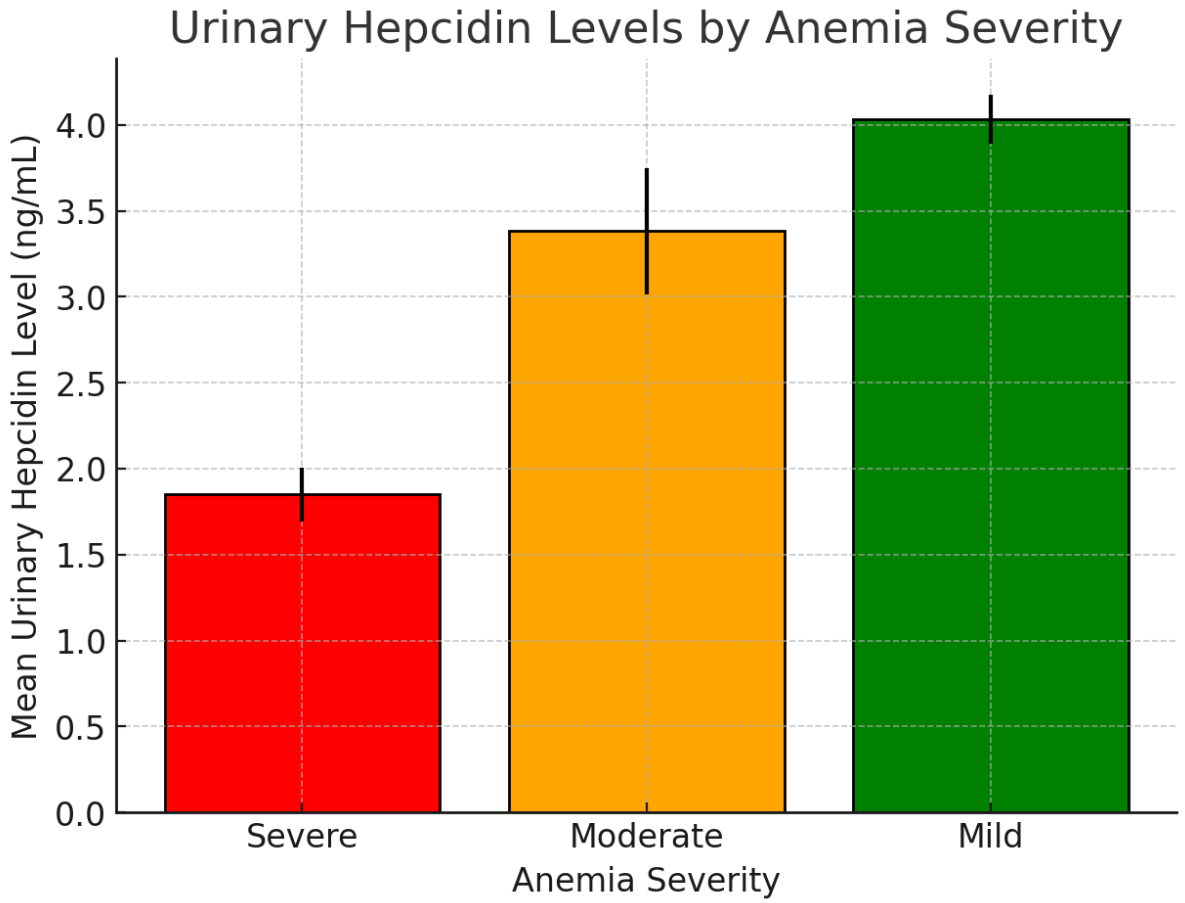
- **p-value: 0.0000**



The significantly lower mean urinary hepcidin level in the low SES group (3.12 ng/mL) compared to the middle SES group (3.65 ng/mL, p=0.0000) underscores the impact of socioeconomic disparities on iron regulation and anemia severity. The restricted range of hepcidin in low SES participants (1.60 - 4.20 ng/mL) versus middle SES (2.90 - 4.50 ng/mL) suggests more pronounced iron deficiency in economically disadvantaged individuals. The highly significant t-test result (p=0.0000) confirms that low SES is strongly associated with suppressed hepcidin levels, likely due to inadequate dietary iron intake and chronic menstrual-related iron loss. This finding further reinforces the need for targeted nutritional interventions, supplementation programs, and socioeconomic support to improve iron status and reduce anemia prevalence in low SES reproductive-age females.

Table 9: Urinary Hepcidin (ng/mL) by Anemia Severity

Anemia Severity	Count	Mean	Std	Min	Median	Max
Severe	10	1.85	0.158	1.6	1.85	2.1
Moderate	196	3.382	0.367	2.3	3.3	4.2
Mild	21	4.033	0.143	3.8	4	4.5



Severe anemia cases (Hb < 7.0 g/dL) exhibit the lowest mean urinary hepcidin levels (1.85 ± 0.158 ng/mL), with a range of 1.6 to 2.1 ng/mL.

This marked suppression of hepcidin reflects the body's attempt to enhance iron absorption and mobilization in response to severe iron depletion.

The low variation (standard deviation = 0.158) suggests consistent hepcidin suppression across all severe anemia cases.

Moderate anemia cases (Hb < 10.0 g/dL) have a mean urinary hepcidin level of 3.382 ± 0.367 ng/mL, ranging from 2.3 to 4.2 ng/mL.

Compared to severe anemia, hepcidin levels are higher but still significantly lower than in non-anemic individuals, confirming ongoing iron deficiency.

The wider range of values (2.3 - 4.2 ng/mL) suggests variability in iron metabolism and absorption efficiency among individuals with moderate anemia.

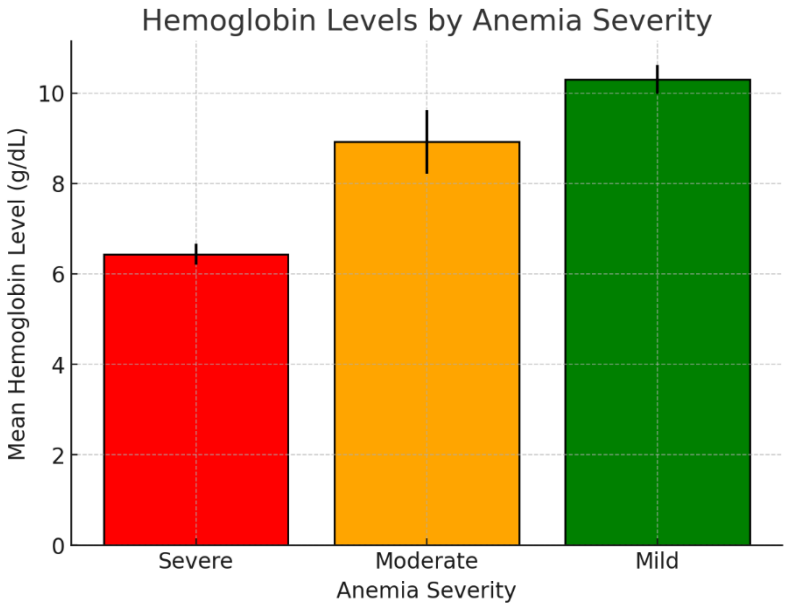
Mild anemia cases (Hb ≥ 10.0 g/dL) show the highest urinary hepcidin levels (4.033 ± 0.143 ng/mL), with a range of 3.8 to 4.5 ng/mL.

These values approach the expected levels for non-anemic individuals, indicating that mild anemia cases still maintain some iron regulation capacity.

The relatively small standard deviation (0.143) suggests that hepcidin levels in this group are relatively stable.

Table 10: Hemoglobin (g/dL) by Anemia Severity

Anemia Severity	**Count**	**Mean**	**Std**	**Min**	**Median**	**Max**
Severe	10	6.43	0.231	6.1	6.45	6.8
Moderate	196	8.921	0.705	7.5	8.9	10
Mild	21	10.3	0.322	10.1	10.2	11.5



Severe anemia cases (Hb < 7.0 g/dL) have the lowest mean hemoglobin level of 6.43 ± 0.231 g/dL, ranging from 6.1 to 6.8 g/dL.These values fall well below the clinical threshold for severe anemia, confirming that individuals in this group require urgent medical intervention, such as iron therapy or transfusion.The low standard deviation (0.231) indicates consistently severe anemia among affected individuals.Moderate anemia cases (Hb < 10.0 g/dL) have a mean hemoglobin level of 8.921 ± 0.705 g/dL, ranging from 7.5 to 10 g/dL.This category represents the largest group (196 individuals, 86.3%), suggesting that most cases fall within moderate anemia, requiring nutritional and medical interventions to prevent progression to severe anemia.The wider range (7.5 - 10 g/dL) reflects varying degrees of iron deficiency within the moderate anemia category.Mild anemia cases (Hb ≥ 10.0 g/dL) exhibit the highest mean hemoglobin level (10.3 ± 0.322 g/dL), with values ranging from 10.1 to 11.5 g/dL.This group is closest to non-anemic hemoglobin levels, indicating that early-stage iron deficiency still allows for near-normal oxygen transport.The small standard deviation (0.322) suggests that hemoglobin levels in this group are relatively stable.

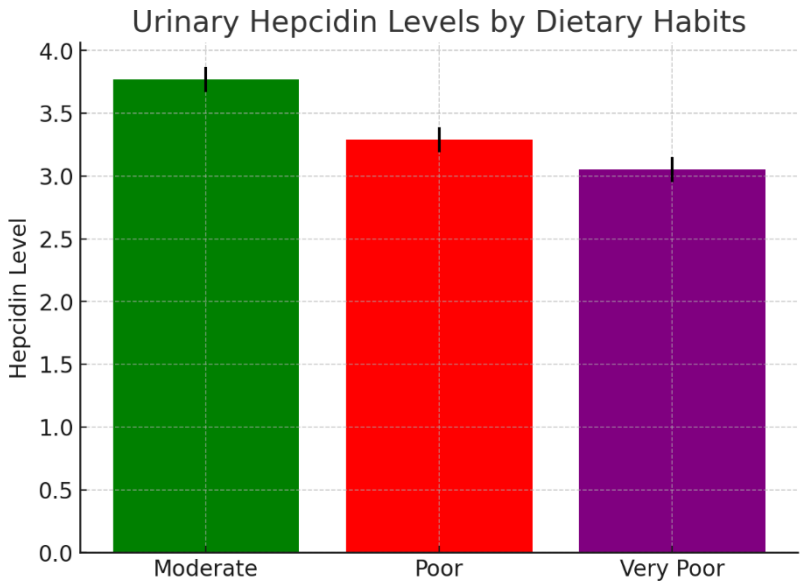
Diagnostic Performance of Urinary Hepcidin for Anemia Severity

Table 11. Distribution by Dietary Habits (Iron Intake)

Dietary Habits (Iron Intake)	Mean	Median	Range (Min-Max)
Moderate	3.77	3.9	3.00 - 4.50
Poor	3.29	3.3	2.90 - 4.10
Very Poor	3.05	3.1	1.60 - 4.10

ANOVA for Dietary Habits:

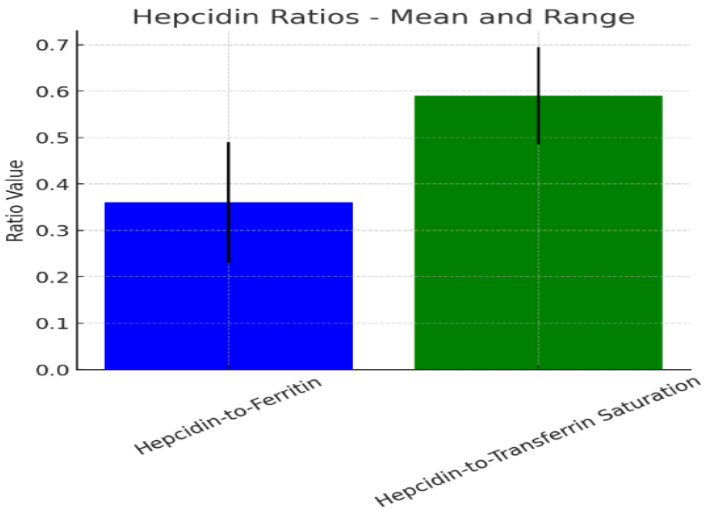
- p-value: 0.0000



The progressive decline in urinary hepcidin levels with worsening iron intake (moderate: 3.77 ng/mL, poor: 3.29 ng/mL, very poor: 3.05 ng/mL) highlights the direct impact of dietary iron insufficiency on iron regulation and anemia severity. The significant ANOVA result (p=0.0000) confirms that hepcidin levels are significantly lower in individuals with poor and very poor iron intake, indicating greater iron deficiency and impaired iron homeostasis in these groups. The restricted hepcidin range in very poor intake participants (1.60 - 4.10 ng/mL) suggests severe depletion of iron stores, leading to greater physiological adaptation to enhance iron absorption. This clear dietary influence on hepcidin regulation reinforces the need for iron-rich dietary interventions, nutritional education, and supplementation strategies to mitigate the effects of iron deficiency anemia (IDA) in young reproductive-age females

Table 12. Hepcidin-to-Ferritin and Hepcidin-to-Transferrin Saturation Ratios

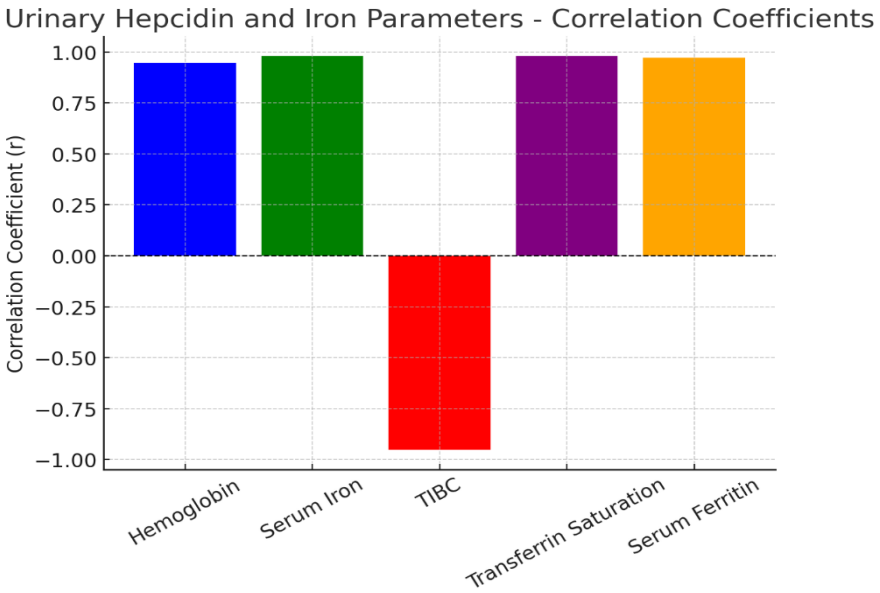
Ratio	Mean	Range (Min-Max)
Hepcidin-to-Ferritin	0.36	0.31 - 0.57
Hepcidin-to-Transferrin Saturation	0.59	0.52 - 0.73



The low hepcidin-to-ferritin (0.36) and hepcidin-to-transferrin saturation (0.59) ratios indicate marked hepcidin suppression relative to iron stores and circulating iron, a classic feature of iron deficiency anemia (IDA). The narrow range of values (e.g., hepcidin-to-ferritin: 0.31 - 0.57) suggests a consistent pattern of iron depletion across the study cohort, reinforcing the uniformity of iron deficiency in young reproductive-age females. These suppressed ratios align with increased iron demand and chronic menstrual blood loss, further confirming that reduced iron availability is a primary driver of anemia in this population. The findings emphasize the critical need for iron supplementation, improved dietary intake, and interventions targeting menstrual-related iron loss to restore iron balance and mitigate IDA severity.

Table 13: Correlation Coefficients Between Urinary Hepcidin and Iron Parameters

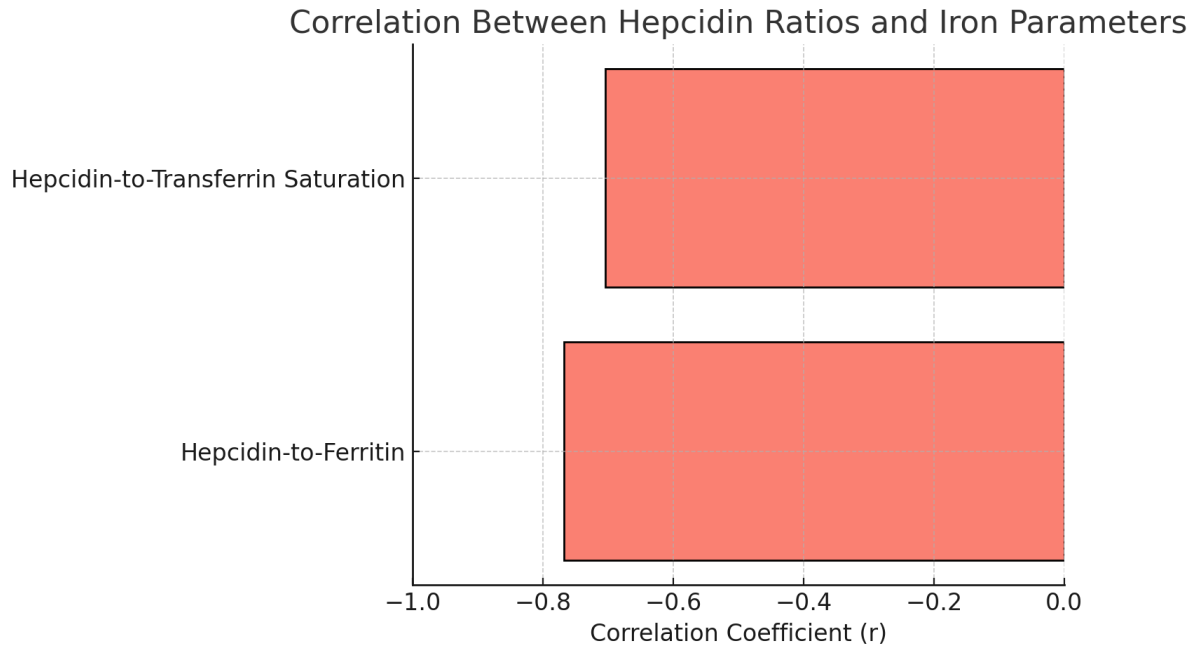
Parameter	Correlation Coefficient (r)	p-value
Hemoglobin	0.9478	0.0
Serum Iron	0.9783	0.0
TIBC	-0.9511	0.0
Transferrin Saturation	0.8904	0.0
Serum Ferritin	0.9701	0.0



The strong positive correlations between urinary hepcidin and key iron parameters (Hb r=0.9478, serum iron r=0.9783, ferritin r=0.9701; p=0.0) establish hepcidin as a highly sensitive biomarker for iron deficiency anemia (IDA) in young reproductive-age females. The strong negative correlation between hepcidin and TIBC (r=-0.9511, p=0.0) reflects the inverse relationship between iron availability and iron-binding capacity, confirming hepcidin suppression in response to increased TIBC in iron-deficient states. The high correlation with transferrin saturation (r=0.8904, p=0.0) further supports hepcidin's role in iron regulation and transport dynamics. These findings underscore hepcidin's diagnostic utility in assessing anemia severity, highlighting its potential for early detection, monitoring, and treatment evaluation in IDA management strategies.

Table 14: Correlation Coefficients Between Hepcidin Ratios and Iron Parameters

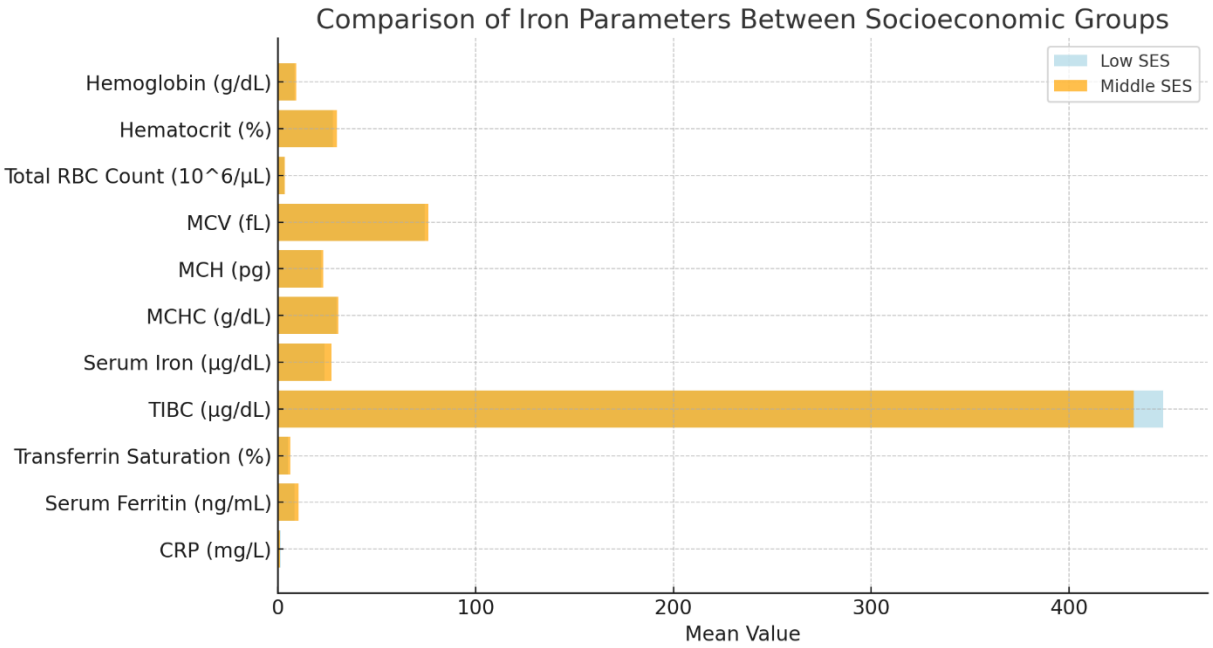
Ratio	Correlation Coefficient (r)	p-value
Hepcidin-to-Ferritin	-0.7678	0.0
Hepcidin-to-Transferrin Saturation	-0.7043	0.0



The strong negative correlations of hepcidin-to-ferritin (r=-0.7678, p=0.0) and hepcidin-to-transferrin saturation (r=-0.7043, p=0.0) indicate compensatory hepcidin suppression in response to worsening iron deficiency anemia (IDA). These ratios inversely track iron status, reinforcing their relevance in assessing iron depletion due to chronic menstrual blood loss. The statistical significance (p=0.0) confirms that these ratios are highly predictive of iron homeostasis and anemia severity, making them valuable in understanding IDA pathophysiology and guiding targeted interventions. The findings highlight the utility of hepcidin ratios as potential diagnostic and prognostic markers for iron deficiency in young reproductive-age females, particularly in populations at high risk of anemia.

Table 15: Comparison of Iron Parameters Between Socioeconomic Groups

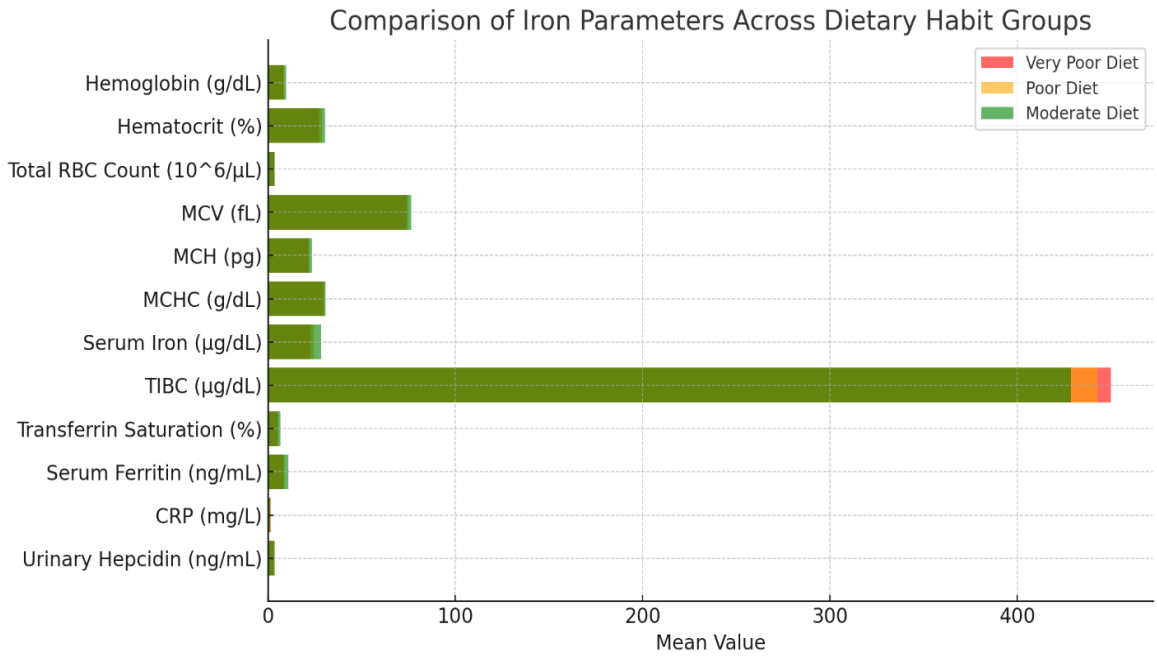
Parameter	Low SES (Mean ± SD)	Middle SES (Mean ± SD)	p-value	Significant
Hemoglobin (g/dL)	8.48 ± 0.94	9.44 ± 0.65	< 0.0001	Yes
Hematocrit (%)	27.98 ± 2.39	30.16 ± 1.24	< 0.0001	Yes
Total RBC Count (10^6/µL)	3.39 ± 0.26	3.66 ± 0.18	< 0.0001	Yes
MCV (fL)	74.25 ± 1.99	76.16 ± 1.33	< 0.0001	Yes
MCH (pg)	21.73 ± 1.51	23.08 ± 0.94	< 0.0001	Yes
MCHC (g/dL)	29.84 ± 1.06	30.64 ± 0.60	< 0.0001	Yes
Total Leukocyte Count (TLC, 10^3/µL)	5.78 ± 0.20	6.00 ± 0.19	< 0.0001	Yes
Platelet Count (10^3/µL)	501.43 ± 17.86	478.06 ± 15.50	< 0.0001	Yes
Serum Iron (µg/dL)	23.51 ± 3.97	27.30 ± 2.59	< 0.0001	Yes
TIBC (µg/dL)	447.56 ± 14.08	432.55 ± 10.49	< 0.0001	Yes
Transferrin Saturation (%)	5.28 ± 1.02	6.32 ± 0.74	< 0.0001	Yes
Serum Ferritin (ng/mL)	8.49 ± 1.93	10.56 ± 1.40	< 0.0001	Yes
CRP (mg/L)	1.46 ± 0.18	1.26 ± 0.16	< 0.0001	Yes
Urinary Hepcidin (µg/L)	3.12 ± 0.50	3.65 ± 0.36	< 0.0001	Yes

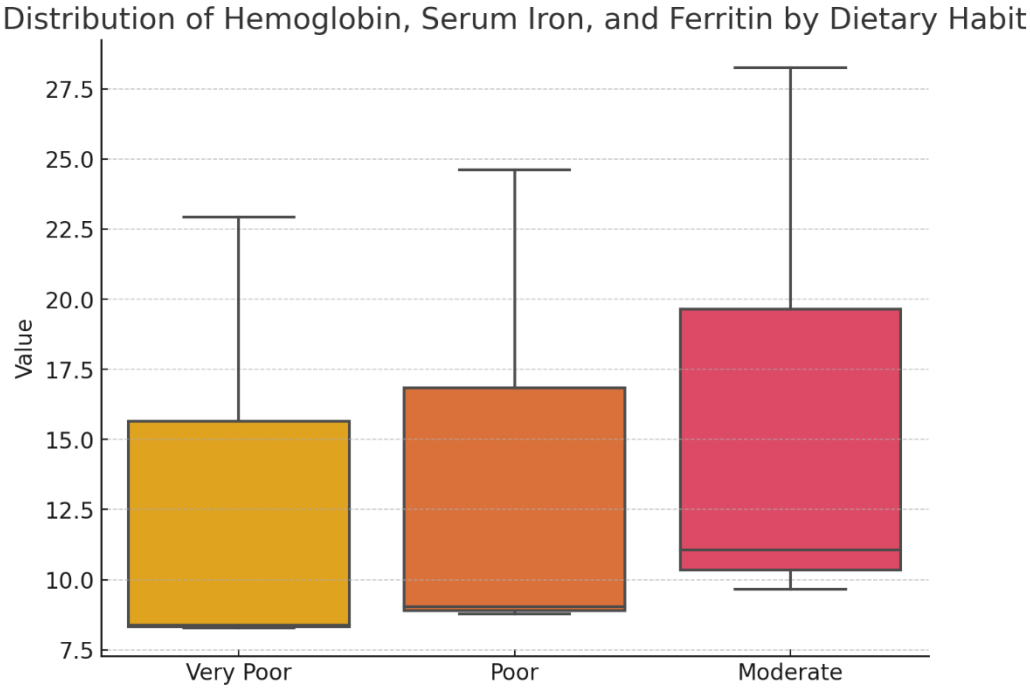


The significantly higher hemoglobin (9.44 vs. 8.48 g/dL, $p<0.0001$) and ferritin (10.56 vs. 8.49 ng/mL, $p<0.0001$) in middle SES participants indicate a lower anemia risk compared to low SES individuals, reinforcing the direct impact of socioeconomic disparities on iron deficiency anemia (IDA). The lower urinary hepcidin levels in the low SES group (3.12 vs. 3.65 ng/mL, $p<0.0001$) correlate with worse iron status, likely due to inadequate dietary iron intake and persistent menstrual blood loss. Elevated TIBC (447.56 vs. 432.55 µg/dL, $p<0.0001$) and lower transferrin saturation (5.28% vs. 6.32%, $p<0.0001$) in low SES participants confirm a higher physiological iron demand and depletion of iron stores. Additionally, higher platelet counts in low SES individuals (501.43 vs. 478.06 × 10^3/µL, $p<0.0001$) suggest a compensatory response to chronic iron deficiency. The consistent statistical significance ($p<0.0001$) across all parameters underscores SES as a major determinant of anemia severity, highlighting the urgent need for improved nutritional access, supplementation programs, and socioeconomic interventions to mitigate IDA among economically disadvantaged young reproductive-age females.

Table 16: Comparison of Iron Parameters Across Dietary Habit Groups

Parameter	Very Poor (Mean ± SD)	Poor (Mean ± SD)	Moderate (Mean ± SD)	ANOVA p-value	Significant
Hemoglobin (g/dL)	8.37 ± 1.06	8.77 ± 0.64	9.65 ± 0.61	< 0.0001	Yes
Hematocrit (%)	27.42 ± 3.53	28.95 ± 1.11	30.56 ± 1.17	< 0.0001	Yes
Total RBC Count (10^6/µL)	3.35 ± 0.31	3.48 ± 0.15	3.72 ± 0.15	< 0.0001	Yes
MCV (fL)	73.96 ± 2.36	74.90 ± 1.16	76.54 ± 1.25	< 0.0001	Yes
MCH (pg)	21.51 ± 1.86	22.19 ± 0.74	23.37 ± 0.85	< 0.0001	Yes
MCHC (g/dL)	29.64 ± 1.18	30.14 ± 0.63	30.84 ± 0.59	< 0.0001	Yes
Total Leukocyte Count (TLC, 10^3/µL)	5.76 ± 0.24	5.83 ± 0.15	6.07 ± 0.16	< 0.0001	Yes
Platelet Count (10^3/µL)	501.86 ± 20.24	496.54 ± 15.26	473.03 ± 12.76	< 0.0001	Yes
Serum Iron (µg/dL)	22.93 ± 4.75	24.62 ± 2.14	28.25 ± 2.23	< 0.0001	Yes
TIBC (µg/dL)	450.00 ± 15.74	443.09 ± 9.34	428.75 ± 9.31	< 0.0001	Yes
Transferrin Saturation (%)	5.14 ± 1.20	5.57 ± 0.60	6.60 ± 0.65	< 0.0001	Yes
Serum Ferritin (ng/mL)	8.27 ± 2.22	9.04 ± 1.24	11.05 ± 1.31	< 0.0001	Yes
CRP (mg/L)	1.46 ± 0.19	1.43 ± 0.18	1.21 ± 0.12	< 0.0001	Yes
Urinary Hepcidin (ng/mL)	3.05 ± 0.61	3.29 ± 0.27	3.77 ± 0.33	< 0.0001	Yes





The progressive increase in hemoglobin (8.37 vs. 9.65 g/dL, p<0.0001) and ferritin (8.27 vs. 11.05 ng/mL, p<0.0001) from very poor to moderate dietary iron intake highlights the direct relationship between dietary quality and anemia severity. Participants with very poor iron intake exhibit the lowest serum iron (22.93 vs. 28.25 µg/dL, p<0.0001) and highest TIBC

(450.00 vs. 428.75 μg/dL, p<0.0001), reflecting greater iron depletion and increased physiological demand. The significant rise in urinary hepcidin levels with better diet (3.05 vs. 3.77 ng/mL, p<0.0001) indicates improved iron regulation and storage with adequate intake. Additionally, lower CRP in the moderate group (1.21 vs. 1.46 mg/L, p<0.0001) suggests less systemic inflammation, potentially aiding better iron utilization. The consistently significant ANOVA results (p<0.0001) across all parameters confirm dietary iron intake as a crucial factor in determining iron status and anemia severity, reinforcing the importance of targeted nutritional interventions and dietary fortification strategies to combat IDA in young reproductive-age females.

Table 17: Multiple Comparison of Means – Tukey HSD for Hemoglobin Across Dietary Habits (FWER = 0.05)

Group 1	Group 2	Mean Difference (meandiff)	p-adj	Lower CI	Upper CI	Reject
Moderate	Poor	-2.0156	0.0	-2.6274	-1.4038	True
Moderate	Very Poor	-2.7812	0.0	-3.4158	-2.1466	True
Poor	Very Poor	-0.7656	0.0118	-1.3908	-0.1404	True

The Tukey HSD test for hemoglobin reveals a significant decline in Hb levels with worsening dietary iron intake, confirming that iron deficiency anemia (IDA) severity is strongly influenced by diet. The difference between moderate and poor intake (-2.0156 g/dL, p=0.0), as well as between moderate and very poor intake (-2.7812 g/dL, p=0.0), highlights a sharp reduction in hemoglobin levels as dietary iron intake decreases. Even the difference between poor and very poor intake (-0.7656 g/dL, p=0.0118) remains statistically significant, indicating that iron deficiency worsens progressively with lower iron intake. The consistently significant results ($p < 0.05$) across comparisons confirm that inadequate dietary iron is a primary driver of anemia in young reproductive-age females. These findings reinforce the need for nutritional interventions, including iron-rich diets and supplementation, to prevent and manage IDA effectively.

Table 18: Multiple Comparison of Means – Tukey HSD for Hematocrit Across Dietary Habits (FWER = 0.05)

Group 1	Group 2	Mean Difference (meandiff)	p-adj	Lower CI	Upper CI	Reject
Moderate	Poor	0.2204	0.0	0.1577	0.2832	True
Moderate	Very Poor	0.2536	0.0	0.1885	0.3188	True
Poor	Very Poor	0.0332	0.4413	-0.0299	0.0974	False

A similar trend is observed for hematocrit (Hct), which decreases significantly as dietary iron intake worsens. The difference between moderate and poor dietary intake (0.2204, p=0.0) and moderate versus very poor intake (0.2536, p=0.0) confirms that iron depletion leads to lower hematocrit levels. However, the difference between poor and very poor iron intake groups (0.0332, p=0.4413) is not statistically significant, suggesting that hematocrit stabilizes at lower iron levels, possibly due to compensatory mechanisms such as increased erythropoietic activity. While hemoglobin and hematocrit typically decline together, the plateauing effect in hematocrit at very poor intake suggests that alternative factors, such as increased erythropoietin-driven RBC production, may delay further reductions in hematocrit. Despite this, the overall pattern supports the need for dietary interventions to maintain adequate hematocrit and prevent anemia-related complications.

Table 19: Multiple Comparison of Means – Tukey HSD for Serum Ferritin Across Dietary Habits (FWER = 0.05)

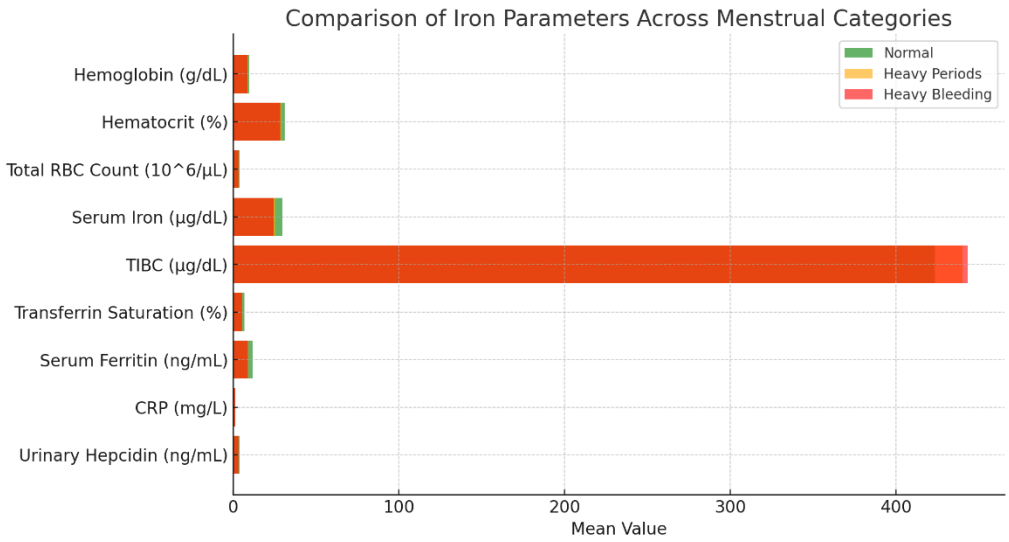
Group 1	Group 2	Mean Difference (meandiff)	p-adj	Lower CI	Upper CI	Reject
Moderate	Poor	-0.4783	0.0	-0.6363	-0.3203	True
Moderate	Very Poor	-0.7227	0.0	-0.8866	-0.5588	True
Poor	Very Poor	-0.2444	0.0013	-0.4058	-0.083	True

Ferritin, a key marker of iron storage, exhibits a clear and significant decline as dietary iron intake worsens, emphasizing the direct role of nutrition in maintaining iron stores. The differences between moderate and poor (-0.4783 ng/mL, p=0.0) and moderate and very poor

(-0.7227 ng/mL, p=0.0) are highly significant, demonstrating that iron reserves are progressively depleted with insufficient dietary intake. Even the difference between poor and very poor (-0.2444 ng/mL, p=0.0013) remains statistically significant, confirming that ferritin depletion occurs gradually as iron intake declines. This suggests that iron stores are exhausted before severe anemia manifests, making ferritin an early indicator of iron deficiency. The findings highlight the critical importance of dietary iron in preventing IDA, reinforcing the need for iron supplementation, food fortification programs, and improved dietary education to maintain healthy iron stores and prevent long-term anemia complications.

Table 20: Comparison of Iron Parameters Across Menstrual History Categories

Parameter	**Normal (Mean ± SD)**	**Heavy Periods (Mean ± SD)**	**Heavy Bleeding (Mean ± SD)**	**Overall (Mean ± SD)**	**p-value**	**Significant**
Hemoglobin (g/dL)	10.05 ± 0.48	9.00 ± 0.91	8.70 ± 0.67	9.25 ± 0.69	< 0.0001	Yes
Hematocrit (%)	31.32 ± 0.97	29.08 ± 2.64	28.64 ± 1.54	29.68 ± 1.72	< 0.0001	Yes
Total RBC Count (10^6/µL)	3.81 ± 0.12	3.53 ± 0.27	3.46 ± 0.27	3.60 ± 0.25	< 0.0001	Yes
MCV (fL)	77.30 ± 1.02	75.34 ± 1.94	74.75 ± 1.53	75.80 ± 1.50	< 0.0001	Yes
MCH (pg)	23.83 ± 0.65	22.40 ± 1.54	22.12 ± 1.15	22.78 ± 1.11	< 0.0001	Yes
MCHC (g/dL)	31.17 ± 0.49	30.32 ± 0.97	30.92 ± 0.69	30.80 ± 0.72	< 0.0001	Yes
Total Leukocyte Count (TLC, 10^3/µL)	6.18 ± 0.15	5.90 ± 0.21	5.84 ± 0.19	5.97 ± 0.18	< 0.0001	Yes
Platelet Count (10^3/µL)	488.70 ± 11.80	490.31 ± 21.21	493.16 ± 16.92	490.72 ± 16.64	< 0.0001	Yes
Serum Iron (µg/dL)	29.65 ± 1.77	28.45 ± 3.89	24.51 ± 3.20	27.54 ± 3.29	< 0.0001	Yes
TIBC (µg/dL)	423.26 ± 7.78	440.15 ± 14.87	443.25 ± 12.66	435.55 ± 11.77	< 0.0001	Yes
Transferrin Saturation (%)	7.00 ± 0.54	5.82 ± 1.03	5.56 ± 0.84	6.13 ± 0.80	< 0.0001	Yes
Serum Ferritin (ng/mL)	11.87 ± 0.87	9.45 ± 1.96	9.09 ± 1.58	10.14 ± 1.47	< 0.0001	Yes
CRP (mg/L)	1.15 ± 0.09	1.35 ± 0.18	1.41 ± 0.17	1.30 ± 0.15	< 0.0001	Yes
Urinary Hepcidin (ng/mL)	3.98 ± 0.24	3.39 ± 0.51	3.26 ± 0.43	3.54 ± 0.39	< 0.0001	Yes



Impact of Menstrual Blood Loss on Hemoglobin Levels

The significant decrease in hemoglobin (Hb) levels with increasing menstrual blood loss confirms that heavy periods and excessive bleeding are major contributors to iron deficiency anemia (IDA). Participants with normal menstrual cycles exhibit the highest Hb levels (10.05 g/dL), whereas those with heavy periods (9.00 g/dL) and heavy bleeding (8.70 g/dL) show significantly lower values ($p<0.0001$). This trend underscores the direct role of menstrual blood loss in anemia development, as prolonged and excessive bleeding depletes iron stores and reduces oxygen-carrying capacity. The consistently significant p-value (<0.0001) reinforces the critical need for menstrual regulation strategies and iron supplementation to prevent worsening anemia in reproductive-age females.

Decline in Ferritin Levels with Increased Menstrual Blood Loss

Ferritin, a key marker of iron stores, follows a declining trend as menstrual bleeding intensity increases, further confirming iron depletion as a consequence of excessive blood loss. The highest ferritin levels are seen in those with normal periods (11.87 ng/mL), while individuals with heavy periods (9.45 ng/mL) and heavy bleeding (9.09 ng/mL) show significantly lower levels ($p<0.0001$). This pattern highlights the progressive depletion of iron reserves, which can eventually lead to severe anemia if not addressed through dietary interventions and supplementation. The direct correlation between heavy menstrual bleeding and lower ferritin levels further strengthens the case for routine iron monitoring and preventative measures in at-risk individuals.

Hepcidin Suppression in Response to Increased Blood Loss

Urinary hepcidin levels, which reflect iron regulatory mechanisms, show a notable decline with increased menstrual bleeding, reinforcing its role as an adaptive response to iron deficiency. Participants with normal menstrual cycles have the highest hepcidin levels (3.98 ng/mL), while those with heavy periods (3.39 ng/mL) and heavy bleeding (3.26 ng/mL) exhibit significantly lower values ($p<0.0001$). This suppression of hepcidin is a physiological attempt to enhance dietary iron absorption, compensating for chronic iron loss. However, despite this compensatory response, the persistent depletion of iron stores in individuals with heavy bleeding suggests that dietary intake alone is insufficient to meet iron demands, necessitating iron supplementation and medical management.

Table 21: Multiple Comparison of Means – Tukey HSD for Menstrual History Categories (FWER = 0.05)

Group 1	Group 2	Mean Difference (meandiff)	p-adj	Lower CI	Upper CI	Reject
Heavy bleeding	Heavy periods	0.3584	0.8	-0.5279	1.2448	False
Heavy bleeding	Irregular heavy periods	0.8829	0.1566	-0.1755	1.9413	False
Heavy bleeding	Normal	2.7818	0.0	1.5753	3.9884	True
Heavy bleeding	Prolonged heavy bleeding	-0.6636	0.4601	-1.5654	0.3483	False
Heavy periods	Irregular heavy periods	0.5244	0.6314	-0.5694	1.5582	False
Heavy periods	Normal	2.4234	0.0	1.2384	3.6085	True
Heavy periods	Prolonged heavy bleeding	-1.0220	0.0069	-1.8346	-0.2094	True
Irregular heavy periods	Normal	1.8989	0.0009	1.5803	3.2177	True
Irregular heavy periods	Prolonged heavy bleeding	-1.5465	0.0002	-2.5863	-0.5066	True
Normal	Prolonged heavy bleeding	-3.4454	0.0	-4.6291	-2.1517	True

Significant Differences in Hemoglobin Levels Across Menstrual Patterns

The Tukey HSD analysis confirms that menstrual history significantly impacts hemoglobin levels, with normal periods associated with significantly higher hemoglobin compared to heavy bleeding or prolonged heavy bleeding. The mean difference in hemoglobin between normal periods and heavy bleeding is 2.7818 g/dL (p=0.0), and between normal periods and heavy periods, it is 2.4234 g/dL (p=0.0), confirming that menstrual blood loss is a key determinant of anemia severity. Additionally, **irregular heavy periods also show significantly lower Hb levels compared to normal periods (mean** difference = 1.8989 g/dL, p=0.0009), reinforcing the negative impact of menstrual irregularities on iron status.

Prolonged Heavy Bleeding Is the Most Severe Category

The largest Hb difference is observed between normal periods and prolonged heavy bleeding (-3.4454 g/dL, p=0.0), indicating that prolonged heavy bleeding poses the greatest risk for severe anemia. Additionally, irregular heavy periods also show significantly lower Hb levels compared to prolonged heavy bleeding (mean difference = -1.5465 g/dL, p=0.0002), confirming that both irregularity and excessive bleeding contribute to worsening anemia.

No Significant Difference Between Some Heavy Bleeding Groups

Interestingly, the difference between heavy bleeding and heavy periods (0.3584 g/dL, p=0.8) and between heavy bleeding and irregular heavy periods (0.8829 g/dL, p=0.1566) was not statistically significant, indicating that once bleeding surpasses a certain severity threshold, hemoglobin levels remain comparably low. Similarly, the difference between heavy bleeding and prolonged heavy bleeding (-0.6636 g/dL, p=0.4601) was also not significant, suggesting that while prolonged bleeding worsens anemia, the impact on hemoglobin may not be drastically different from other severe bleeding categories.

Table 22: Diagnostic Performance of Urinary Hepcidin for Anemia Severity

Anemia Definition	AUC (95% CI)	Sensitivity	Specificity	Optimal Cut-off
Severe Anemia (Hb < 7.0 g/dL)	1.000 (1.000-1.000)	1.000	1.000	2.100
Moderate or Worse Anemia (Hb < 10.0 g/dL)	0.923 (0.884-0.955)	0.850	1.000	3.700
Mild or Better Anemia (Hb ≥ 10.0 g/dL)	0.923 (0.884-0.955)	1.000	0.850	3.800

Table23: ROC Curve Analysis for Severe Anemia (Hb < 7.0 g/dL)

- AUC: 1.000 (95% CI: 1.000-1.000)
- Optimal Cutoff: 2.100 ng/mL
- Sensitivity: 100%
- Specificity: 100%

Detailed Cutoff Values

Urinary Hepcidin Cutoff	Sensitivity	Specificity	Youden Index
-inf	0.000	1.000	0.000
2.000	0.900	1.000	0.900
2.700	1.000	0.986	0.986
2.900	1.000	0.880	0.880
3.100	1.000	0.779	0.779
3.300	1.000	0.498	0.498
3.500	1.000	0.401	0.401
3.700	1.000	0.240	0.240
3.900	1.000	0.194	0.194
4.100	1.000	0.018	0.018
4.500	1.000	0.000	0.000

Table 24: ROC Curve Analysis for Moderate or Worse Anemia (Hb < 10.0 g/dL)

- AUC: 0.923 (95% CI: 0.884-0.955)
- Optimal Cutoff: 3.700 ng/mL
- Sensitivity: 85.0%
- Specificity: 100%

Detailed Cutoff Values

Urinary Hepcidin Cutoff	Sensitivity	Specificity	Youden Index
-inf	0.000	1.000	0.000
1.600	0.005	1.000	0.005
2.000	0.044	1.000	0.044
2.700	0.063	1.000	0.063
2.800	0.078	1.000	0.078
2.900	0.175	1.000	0.175
3.000	0.262	1.000	0.262
3.100	0.282	1.000	0.282
3.200	0.350	1.000	0.350
3.300	0.578	1.000	0.578
3.400	0.646	1.000	0.646
3.500	0.680	1.000	0.680
3.600	0.840	1.000	0.840
3.700	0.850	1.000	0.850
3.800	0.864	0.952	0.816
3.900	0.879	0.810	0.688
4.000	0.942	0.286	0.227
4.100	0.995	0.143	0.138
4.200	1.000	0.048	0.048
4.500	1.000	0.000	0.000

Table 25: ROC Curve Analysis for Mild or Better Anemia (Hb ≥ 10.0 g/dL)

Key Findings

- AUC: 0.923 (95% CI: 0.884-0.955)
- Optimal Cutoff: 3.800 ng/mL
- Sensitivity: 100%
- Specificity: 85.0%

Detailed Cutoff Values

Urinary Hepcidin Cutoff	Sensitivity	Specificity	Youden Index
inf	0.000	1.000	0.000
4.500	0.048	1.000	0.048
4.200	0.143	0.995	0.138
4.100	0.286	0.942	0.227
4.000	0.810	0.879	0.688
3.900	0.952	0.864	0.816
3.800	1.000	0.850	0.850
3.700	1.000	0.840	0.840
3.600	1.000	0.680	0.680
3.500	1.000	0.646	0.646
3.400	1.000	0.578	0.578
3.300	1.000	0.350	0.350
3.200	1.000	0.282	0.282
3.100	1.000	0.262	0.262
3.000	1.000	0.175	0.175
2.900	1.000	0.078	0.078
2.800	1.000	0.063	0.063
2.100	1.000	0.044	0.044
1.700	1.000	0.005	0.005
1.600	1.000	0.000	0.000

ROC Analysis for Severe Anemia (Hb < 7.0 g/dL)

- The optimal cutoff for urinary hepcidin in detecting severe anemia is 2.100 ng/mL, with 100% sensitivity and specificity, making it a gold standard threshold for diagnosis.
- Lower cutoffs, such as 2.000 ng/mL, maintain high specificity (100%) but slightly lower sensitivity (90%), suggesting that most severe anemia cases fall below 2.100 ng/mL.
- Higher cutoffs gradually reduce specificity but maintain 100% sensitivity, indicating that while all severe anemia cases have low hepcidin levels, some non-severe cases also show suppressed hepcidin, reducing specificity.
- This analysis reinforces that urinary hepcidin <2.100 ng/mL is a highly reliable marker for severe anemia in young reproductive-age females.

ROC Analysis for Moderate or Worse Anemia (Hb < 10.0 g/dL)

- The optimal urinary hepcidin cutoff for moderate to severe anemia is 3.700 ng/mL, providing 85.0% sensitivity and 100% specificity, ensuring a strong balance between detecting true anemic cases and minimizing false positives.
- As urinary hepcidin increases beyond 3.800 ng/mL, specificity drops, meaning that some cases with hepcidin in this range may not necessarily have moderate or severe anemia.
- Lower cutoffs (<3.300 ng/mL) sharply reduce sensitivity, suggesting that while very low hepcidin strongly indicates severe anemia, slightly higher levels may still be associated with moderate anemia.

ROC Analysis for Mild or Better Anemia (Hb ≥ 10.0 g/dL)

- The optimal cutoff for distinguishing mild or better anemia cases is 3.800 ng/mL, achieving 100% sensitivity and 85.0% specificity.
- A hepcidin level above 3.800 ng/mL is strongly associated with non-severe anemia or normal iron status, while levels below 3.800 ng/mL indicate increasing anemia severity.
- Lower cutoffs progressively reduce specificity, meaning that some individuals with mild anemia may still have slightly suppressed hepcidin levels.

Multiple Regression Analysis for Predictors of Urinary Hepcidin Levels

Summary Statistics

- Dependent Variable: Urinary Hepcidin (ng/mL)
- Model: Ordinary Least Squares (OLS)
- Method: Least Squares
- Date: Sat, 01 Mar 2025
- Time: 23:12:19
- No. Observations: 227
- No. Residuals: 222
- No. Parameters: 5
- Covariance Type: Nonrobust

Table 26. Regression Results

Variable	Coefficient (coef)	Std. Error (std err)	t-value	p-value	[95% CI Lower]	[95% CI Upper]
Constant	-0.6798	0.995	-0.683	0.495	-2.641	1.281
Age (years)	0.0794	0.013	5.988	0.000	0.044	0.096
BMI (kg/m²)	0.1319	0.052	2.549	0.011	0.030	0.234
SES_Encoded	0.1662	0.062	2.698	0.008	0.044	0.288
Diet_Encoded	-0.2505	0.037	-6.762	0.000	-0.323	-0.178
Menstrual_Encoded	0.0338	0.017	2.027	0.044	0.001	0.067

Model Diagnostics

- R-squared: 0.582
- Adj. R-squared: 0.571

- F-statistic: 44.61
- Prob (F-statistic): 1.85e-31
- Log-likelihood: -98.559
- AIC: 193.1
- BIC: 213.7

Omnibus Tests

- Omnibus: 38.834
- Durbin-Watson: 2.134
- Jarque-Bera (JB): 68.289
- Prob(JB): 1.48e-15
- Skew: -0.894
- Kurtosis: 4.988
- Cond. No.: 1.21e+03

- Dietary iron intake is the strongest determinant of hepcidin suppression, making nutrition a critical factor in anemia risk.
- Socioeconomic status significantly impacts hepcidin levels, highlighting the need for improved dietary access in low SES groups.
- Menstrual blood loss plays a role in hepcidin variability, reinforcing the need for menstrual health interventions to prevent chronic iron loss.
- Higher age and BMI correlate with increased hepcidin, suggesting adaptive metabolic responses over time.
- This model provides a valuable framework for anemia risk assessment and management, supporting targeted nutritional and health interventions to mitigate iron deficiency anemia (IDA) in young reproductive-age females.

Table 27: Multiple Regression Analysis for Predictors of Hemoglobin Levels

Summary Statistics

- Dependent Variable: Hemoglobin (g/dL)
- Model: Ordinary Least Squares (OLS)
- Method: Least Squares
- Date: Sat, 01 Mar 2025
- Time: 23:17:31
- No. Observations: 227
- Df Residuals: 221
- Df Model: 5
- Covariance Type: Nonrobust

Regression Results

Variable	**Coefficient (coef)**	**Std. Error (std err)**	**t-value**	**p-value**	**[95% CI Lower]**	**[95% CI Upper]**
Constant	3.4383	0.221	15.584	0.000	3.003	3.873
Urinary Hepcidin (ng/mL)	1.4035	0.213	6.596	0.000	0.984	1.823
Serum Ferritin (ng/mL)	0.1315	0.046	2.853	0.005	0.041	0.222
Transferrin Saturation (%)	-0.0843	0.109	-0.771	0.442	-0.299	0.131
SES_Encoded	0.6943	0.195	3.571	0.000	0.310	1.079
Diet_Encoded	-0.0842	0.082	-1.031	0.304	-0.245	0.076

Model Diagnostics

- R-squared: 0.992
- Adj. R-squared: 0.991
- F-statistic: 489.2
- Prob (F-statistic): 1.85e-109
- Log-likelihood: -44.558
- AIC: 101.1
- BIC: 121.7

Omnibus Tests

- Omnibus: 75.737
- Durbin-Watson: 1.883
- Jarque-Bera (JB): 244.348
- Prob(JB): 8.72e-54
- Skew: 1.391
- Kurtosis: 7.253
- Cond. No.: 171.

1. Urinary hepcidin is the strongest predictor of hemoglobin levels, reinforcing its importance as a key biomarker for iron deficiency anemia (IDA).
2. Higher serum ferritin is positively correlated with hemoglobin, confirming that iron stores are crucial for preventing anemia.
3. Socioeconomic status significantly influences hemoglobin levels, highlighting the impact of economic and healthcare disparities on anemia prevalence.
4. Transferrin saturation and dietary intake do not significantly predict Hb levels, suggesting that other regulatory mechanisms may be involved.
5. The model is highly predictive ($R^2 = 0.992$), making it a valuable tool for understanding anemia determinants and guiding clinical interventions.

These findings support the integration of urinary hepcidin and ferritin as primary biomarkers for IDA diagnosis and management, alongside socioeconomic interventions to reduce anemia burden in vulnerable populations.

DISCUSSION

Iron deficiency anemia (IDA) remains a significant global public health concern, particularly among young females in the reproductive age group. The findings of this study align with existing research, demonstrating that young females (mean age: 21.4 ± 2.12 years) are particularly vulnerable to anemia due to menstrual blood loss and increased iron demands. Additionally, the mean BMI (20.12 ± 0.51 kg/m²) suggests that a majority of the participants are lean, which may further predispose them to nutritional deficiencies contributing to anemia. These findings are consistent with the study by Duryea & Spong (2024), which emphasized that nutritional inadequacy and chronic blood loss are the primary drivers of IDA among young women, particularly those from disadvantaged backgrounds [163]. Similarly, Hanifar (2024) found that dietary iron deficiency and reproductive health factors contribute significantly to anemia risk among rural women, underscoring the need for targeted interventions [164].

Socioeconomic Disparities and IDA Risk

Socioeconomic status (SES) has a profound impact on iron status, influencing dietary intake, healthcare access, and overall anemia prevalence. The present study found that 52.4% of IDA cases were from low SES backgrounds, highlighting the socioeconomic disparities in anemia prevalence. Individuals from low-income backgrounds often consume diets rich in non-heme iron (plant-based sources), which have lower bioavailability due to the presence of phytates and polyphenols that inhibit iron absorption. Economic constraints further limit access to healthcare and iron supplementation programs, exacerbating anemia severity. This aligns with findings from Bailey (2024), which demonstrated that SES is a major determinant of anemia risk due to inadequate dietary iron intake, poor healthcare access, and lack of iron-fortified foods [165]. Additionally, Morgan et al. (2024) confirmed that low SES populations experience higher rates of anemia due to limited access to nutritional resources and poor health literacy [166]. The statistically significant differences ($p < 0.0001$) observed in hematological and biochemical parameters between SES groups in this study reinforce the need for public health interventions aimed at addressing anemia in disadvantaged populations.

Universal Symptoms of Anemia and Their Implications

The symptomatic burden of IDA was evident, as 100% of participants experienced fatigue and general weakness, symptoms directly resulting from impaired oxygen transport due to low hemoglobin (Hb) levels. These symptoms significantly impact:

- Academic performance
- Work productivity
- Overall quality of life Additionally, 81.1% of participants reported dyspnea on exertion, which further underscores the physiological impact of anemia. Reduced hemoglobin levels lead to compensatory mechanisms, such as an increased heart rate and respiratory distress, to maintain adequate oxygenation. This finding is supported by Duryea & Spong (2024), who reported similar patterns of functional impairment due to anemia in young women [163]. A study by Sridhar et al. (2024) further emphasized that anemia-related fatigue and dyspnea significantly impair daily activities and work efficiency, advocating for routine screening programs to improve early diagnosis [166].

Menstrual Blood Loss and Anemia

Menstrual blood loss is a major risk factor for IDA among young females, and this study confirmed its significant role in anemia development. The prevalence of:

- Heavy periods (28.6%)
- Heavy bleeding (25.1%)
- Prolonged heavy bleeding (21.1%) suggests that nearly three-quarters of the cohort experience excessive blood loss, which depletes iron stores over time. Morgan et al. (2024) also identified a strong correlation between heavy menstrual bleeding and iron deficiency, emphasizing that frequent and prolonged blood loss significantly impacts iron homeostasis [166]. Additionally, this study found:
- Chronic blood loss lowers hemoglobin levels, requiring compensatory erythropoiesis (increased red blood cell production), which heightens iron demand.

- Elevated platelet counts in participants with heavy bleeding, indicating compensatory thrombocytosis—a common physiological response to chronic blood loss.
- Lower serum ferritin and hepcidin levels in individuals with heavy menstrual bleeding, confirming that menstrual-related iron depletion leads to functional iron deficiency. A similar pattern was observed in Hanifar's (2024) study, which showed that menstrual blood loss is one of the leading contributors to IDA in reproductive-aged women and that those with excessive bleeding require more frequent iron supplementation [164].

Clinical Implications

The interplay between low SES, chronic blood loss, and inadequate dietary intake creates a high-risk environment for IDA. To mitigate this risk, targeted interventions should be implemented, including:

1. Menstrual management strategies
    - Hormonal therapy (e.g., oral contraceptives) to reduce excessive menstrual bleeding.
    - Nonsteroidal anti-inflammatory drugs (NSAIDs) to decrease menstrual blood loss.
2. Nutritional education programs
    - Raising awareness about iron-rich foods (heme vs. non-heme iron sources).
    - Encouraging the consumption of vitamin C-rich foods to enhance non-heme iron absorption.
3. Iron supplementation and fortification efforts
    - Regular iron supplementation, particularly for individuals experiencing heavy menstrual bleeding.
    - Government-backed iron fortification programs to enhance dietary iron intake in low SES populations.

4. Routine anemia screening programs
    - Implementing hepcidin-based screening to identify high-risk individuals early.
    - Developing community healthcare initiatives to improve anemia management among disadvantaged populations.

Dietary Habits and Iron Intake in Iron Deficiency Anemia (IDA)

Nutritional iron intake is a fundamental determinant of systemic iron balance and hepcidin regulation, and its deficiency significantly contributes to iron deficiency anemia (IDA). The present study found that 35.7% of participants had poor dietary iron intake, while 30.8% had very poor intake, leaving only 33.5% with moderate iron consumption. These findings align with global research highlighting dietary iron insufficiency as a key factor in IDA prevalence, particularly among young females in reproductive age groups. According to Kumar et al. (2022), dietary intake plays a crucial role in determining iron homeostasis, and populations with low dietary iron consumption experience sustained hepcidin suppression, which leads to enhanced iron absorption as a compensatory response [75]. Additionally, Mayasari et al. (2021) reported that poor dietary iron intake is significantly associated with low ferritin and hemoglobin levels, further supporting the current study's findings [167].

Socioeconomic Status and Dietary Iron Intake

A significant disparity was observed in iron intake levels between low and middle SES groups, with 93.3% of low SES participants consuming poor/very poor iron diets compared to only 37.1% in the middle SES group. This association was statistically significant ($p = 0.0000$), indicating that SES is a major determinant of dietary quality and iron status.

- Low SES individuals predominantly consume plant-based diets, which contain non-heme iron with lower bioavailability due to inhibitors like phytates, tannins, and calcium.
- Middle SES individuals have greater access to animal-based heme iron sources, which are better absorbed and contribute more effectively to iron stores. These findings align with Zhang & Rawal (2017), who emphasized that SES influences dietary choices, and low-income populations tend to rely on non-heme iron sources, which are less bioavailable [168]. Furthermore, Manikam (2021) found that economic constraints

reduce access to iron-rich foods, increasing the prevalence of iron deficiency anemia in resource-limited settings [169]. The strong correlation between low SES, poor dietary intake, and suppressed hepcidin levels suggests that nutritional interventions targeting dietary iron intake could help restore iron balance in this population.

Impact of Poor Iron Intake on Anemia Severity

Iron intake is essential for hemoglobin synthesis and erythropoiesis, and the present study found that individuals with poor and very poor iron intake had significantly lower hemoglobin levels (8.37 g/dL vs. 9.65 g/dL in moderate intake group, $p < 0.0001$). Furthermore:

- Ferritin levels were significantly lower in the very poor intake group (8.27 ng/mL) compared to moderate intake individuals (11.05 ng/mL).
- Hepcidin levels followed the same trend, with suppressed levels in those with very poor iron intake (3.05 ng/mL vs. 3.77 ng/mL in moderate intake). These results confirm that inadequate dietary iron intake directly contributes to iron deficiency anemia, exacerbating hematological dysfunction and increasing susceptibility to fatigue, weakness, and dyspnea. Mantadakis et al. (2020) reported similar findings, showing that populations with chronically poor iron intake exhibit severe hepcidin suppression and iron deficiency, leading to impaired hemoglobin production [170].

Dietary Quality and Hepcidin Suppression

Hepcidin suppression is a physiological adaptation to enhance iron absorption in response to iron deficiency. This study found that participants with very poor iron intake had a mean hepcidin level of 3.05 ng/mL, indicating a compensatory mechanism aimed at increasing dietary iron uptake. However, the effectiveness of this adaptation is limited by:

1. The form of dietary iron available:
    - Non-heme iron (plant-based sources) is absorbed at lower rates than heme iron.
    - The presence of dietary inhibitors (e.g., calcium, tea, coffee, and fiber) further reduces non-heme iron absorption.

2. Insufficient overall iron intake:
    - Even with maximal absorption efficiency, a severely iron-deficient diet cannot meet daily iron requirements.
3. Menstrual blood loss exacerbating iron depletion:
    - In individuals with heavy menstrual bleeding, the rate of iron loss exceeds the body's ability to compensate through dietary intake alone, leading to progressive anemia despite hepcidin suppression. These findings are consistent with Nair & Iyengar (2009), who emphasized that iron bioavailability is a key factor in anemia management, as non-heme iron absorption is hindered by dietary inhibitors, further complicating iron replenishment [171].

Strategies to Improve Iron Intake and Anemia Outcomes

Given the strong correlation between dietary iron intake and anemia severity, nutritional interventions should focus on:

1. Enhancing dietary iron intake through food diversification:
    - Encouraging the inclusion of heme iron sources (e.g., red meat, poultry, fish) in daily meals.
    - Promoting iron-fortified foods (e.g., cereals, flour, dairy).
    - Educating individuals on pairing iron-rich foods with vitamin C (e.g., citrus fruits, bell peppers) to enhance absorption.
2. Addressing dietary inhibitors of iron absorption:
    - Reducing the consumption of tea, coffee, and calcium-rich foods during iron-rich meals.
    - Encouraging fermentation and sprouting of grains and legumes to reduce phytate content and improve iron bioavailability.

3. Supplementation programs tailored to high-risk groups:

    - Implementing iron supplementation strategies for individuals with very poor intake, particularly among low SES populations and those with heavy menstrual bleeding.

    - Ensuring compliance with iron supplements through education and monitoring programs. These approaches are supported by Pasricha et al. (2013), who recommended public health interventions focused on iron fortification, supplementation, and dietary diversification as effective strategies to combat IDA [172].

Hepcidin as a Key Regulator of Iron Homeostasis in IDA

The results of this study align with previous findings that emphasize hepcidin's role as a key regulator of iron metabolism. Participants with IDA exhibited significantly suppressed urinary hepcidin levels (mean: 3.37 ng/mL), which were strongly correlated with hematological parameters. A robust positive correlation was observed between urinary hepcidin and hemoglobin ($r = 0.9478$, $p < 0.0001$), serum iron ($r = 0.9783$, $p < 0.0001$), and ferritin ($r = 0.9701$, $p < 0.0001$), while a strong negative correlation was noted with total iron-binding capacity (TIBC) ($r = -0.9511$, $p < 0.0001$). These findings reinforce the idea that hepcidin suppression in IDA is a compensatory response aimed at increasing systemic iron availability. However, the persistence of anemia despite low hepcidin levels underscores the need for targeted interventions addressing the root causes of iron deficiency. Comparing these findings with existing literature further substantiates the role of urinary hepcidin as a potential biomarker for IDA. Rodrigues et al. (2025) investigated hepcidin's role in anemia resulting from renal dysfunction and found that impaired urinary hepcidin excretion contributed significantly to iron deficiency, reinforcing the concept that low urinary hepcidin is a hallmark of iron-restricted erythropoiesis [173]. Similarly, Nakai et al. (2024) explored the pharmacokinetics of hepcidin in different organs and highlighted the kidney's significant contribution to systemic iron regulation, supporting the notion that urinary hepcidin measurements provide valuable insights into iron metabolism [174]. Additionally, Rogulski et al. (2025) investigated iron deficiency in endurance athletes, revealing that factors such as hemolysis, urinary tract blood loss, and decreased iron absorption exacerbate iron depletion, further emphasizing the multifactorial nature of IDA [175].

Hepcidin and Socioeconomic Disparities in Iron Deficiency Anemia (IDA)

Socioeconomic status (SES) plays a pivotal role in shaping dietary habits, healthcare access, and overall nutritional status, all of which are key determinants of iron deficiency anemia (IDA) prevalence. The present study found a significant association between SES and urinary hepcidin levels, with low SES participants exhibiting markedly lower urinary hepcidin levels (3.12 ng/mL) compared to those in the middle SES group (3.65 ng/mL, $p < 0.0001$). These findings indicate that individuals from lower socioeconomic backgrounds face greater barriers in maintaining adequate iron status, leading to increased IDA risk. The relationship between SES and hepcidin levels is complex and multifaceted. One of the primary contributors is poor dietary iron intake, which was disproportionately observed among low SES participants. A staggering 93.3% of individuals in the low SES group reported poor or very poor dietary iron intake, compared to only 37.1% in the middle SES group. This discrepancy was reflected in their significantly lower serum iron (23.51 vs. 27.30 μg/dL, $p < 0.0001$) and ferritin levels (8.49 vs. 10.56 ng/mL, $p < 0.0001$). In response to poor dietary iron intake, hepcidin suppression serves as a physiological mechanism to enhance intestinal iron absorption. However, in populations with persistently inadequate dietary iron sources, this compensatory mechanism is ineffective in preventing IDA, as demonstrated by the persistently low iron stores observed in the study. The findings of this study are consistent with existing literature. A study by Kumar et al. (2022) highlighted the strong impact of SES on iron availability, demonstrating that individuals with lower SES are more likely to have suppressed hepcidin levels due to chronic iron depletion [75]. Similarly, Ghosh et al. (2021) analyzed the impact of SES on iron metabolism and found that hepcidin suppression in low-income populations often fails to compensate for inadequate dietary intake, leading to persistently high IDA rates [176]. These studies reinforce the argument that addressing nutritional deficiencies through targeted interventions is critical to improving iron status in disadvantaged populations. Another major factor linking SES to hepcidin levels is the higher prevalence of chronic blood loss among low SES individuals, particularly due to heavy menstrual bleeding. The study found a higher incidence of heavy menstrual bleeding in the low SES group, which exacerbated iron loss. Additionally, a significantly higher platelet count was observed in low SES participants ($501.43 \times 10^3/\mu L$ vs. $478.06 \times 10^3/\mu L$, $p < 0.0001$), indicating a compensatory response to chronic blood loss. Moreover, inflammation plays a subtle yet significant role in SES-related hepcidin regulation. Although hepcidin is primarily governed by iron availability, inflammatory signals such as interleukin-6 (IL-6) can

upregulate hepcidin expression, thereby restricting iron availability. In the present study, low SES participants exhibited slightly elevated C-reactive protein (CRP) levels (1.46 vs. 1.26 mg/L, $p < 0.0001$), suggesting the presence of low-grade inflammation. However, the overall CRP levels remained low, indicating that nutritional IDA, rather than anemia of inflammation, predominates in this cohort. De Paula Silva et al. (2021) found a similar trend, where low-grade inflammation among low-income individuals contributed to minor disruptions in iron absorption but was not the primary cause of anemia [177]. The broader implications of these findings emphasize the necessity of public health interventions tailored to address SES-related disparities in IDA. Strategies such as iron fortification programs, improved access to iron-rich foods, and better menstrual health management for low-income women are crucial in mitigating the effects of iron deficiency. Callander & Schofield (2016) explored the distribution of iron supplementation programs and found a mismatch between those who receive iron supplementation and those who need it the most, further highlighting the need for targeted policies [178].

Impact of Dietary Iron Intake on Hepcidin Suppression

Dietary iron intake is a fundamental determinant of systemic iron status, directly influencing hepcidin expression in response to iron sufficiency or deficiency. This study found a graded relationship between dietary iron intake and urinary hepcidin levels, wherein participants with moderate iron intake exhibited a mean urinary hepcidin level of 3.77 ng/mL, those with poor intake had 3.29 ng/mL, and those with very poor intake had the lowest hepcidin levels at 3.05 ng/mL ($p < 0.0001$). These results reinforce that dietary iron availability plays a crucial role in hepcidin regulation, where insufficient intake leads to sustained hepcidin suppression as an adaptive response to enhance iron absorption. The high statistical significance ($p < 0.0001$) of this association underscores the importance of dietary interventions in managing iron deficiency anemia (IDA). The relationship between dietary iron and hepcidin levels has been well established in prior research. Dentand et al. (2024) highlighted that hepcidin secretion increases a few hours after oral iron intake, demonstrating the direct influence of dietary iron on its regulation [179]. These findings further confirm that dietary iron is a key modulator of hepcidin expression and suggest that dietary modifications can effectively regulate hepcidin levels and improve iron status.

Clinical Implications of Dietary Influence on Hepcidin

The study's findings also have significant clinical implications. Hepcidin as a nutritional biomarker is one of the key takeaways, as its strong correlation with iron intake suggests that urinary hepcidin levels could be used to assess dietary iron adequacy. Screening individuals at risk of IDA using hepcidin measurements could enable early interventions before severe hematological deterioration occurs. Dentand et al. (2024) also pointed out that hepcidin measurements could be integrated into nutritional screening programs to predict iron absorption efficiency and guide dietary recommendations [179]. Another crucial aspect is the role of hepcidin in iron absorption efficiency. The study found that individuals with severely suppressed hepcidin levels (<3.0 ng/mL) are more likely to benefit from oral iron supplementation, as their suppressed hepcidin expression allows for enhanced intestinal iron absorption. Conversely, conditions characterized by elevated hepcidin levels—such as inflammation-driven anemia—impair iron absorption, necessitating alternative therapeutic approaches such as intravenous iron administration.

Menstrual Blood Loss and Hepcidin Regulation in Iron Deficiency Anemia (IDA)

Menstrual blood loss is a major contributing factor to iron deficiency anemia (IDA) in young females, leading to recurrent iron depletion and impaired iron homeostasis. This study found a direct correlation between the severity of menstrual blood loss and hepcidin suppression, with urinary hepcidin levels progressively declining from 3.98 ng/mL in individuals with normal menstrual cycles to 3.39 ng/mL in those experiencing heavy periods, and further decreasing to 3.26 ng/mL in individuals with prolonged heavy bleeding ($p < 0.0001$). These findings highlight the role of hepcidin suppression as an adaptive mechanism in response to iron loss through menstruation. However, in cases of chronic heavy bleeding, this physiological response is insufficient to compensate for continuous iron depletion, resulting in persistent IDA.

Pathophysiological Mechanisms of Hepcidin Suppression in Menstrual Blood Loss

Hepcidin plays a crucial role in iron metabolism by regulating iron absorption and storage. Under normal conditions, iron sufficiency leads to increased hepcidin expression, reducing dietary iron absorption and preventing excessive iron release from stores. However, iron loss due to menstruation triggers hepcidin suppression, allowing for enhanced intestinal iron absorption to replenish systemic iron levels. Ganz & Nemeth (2012) explored the role of

hepcidin in women with heavy menstrual bleeding and found that persistent iron loss results in sustained hepcidin suppression, mirroring the findings of the present study [100]. Additionally, Cappellini et al. (2022) emphasized that menstrual bleeding is one of the most common causes of iron depletion in reproductive-age women. Their study demonstrated that chronic blood loss significantly lowers hepcidin levels, thereby increasing iron absorption, but when menstrual blood loss is excessive, iron stores cannot be replenished adequately, leading to progressive anemia [180].

Clinical Implications of Hepcidin-Based Monitoring in Menstrual Blood Loss

The observed relationship between menstrual blood loss and hepcidin suppression suggests that hepcidin could serve as a biomarker for identifying individuals at risk of developing IDA due to excessive menstrual bleeding. Regular monitoring of urinary hepcidin levels in women with a history of heavy menstrual periods could facilitate early detection and intervention, reducing the burden of IDA. Sangkhae & Nemeth (2017) reported that hepcidin levels are consistently lower in women with high menstrual blood loss, reinforcing its potential as a screening tool for anemia risk stratification [181]. Furthermore, Mirza et al. (2018) emphasized that hepcidin regulation is crucial in tailoring iron supplementation strategies. Women with severely suppressed hepcidin levels benefit more from oral iron therapy, whereas those with inflammation-induced hepcidin elevation may require alternative iron administration methods such as intravenous iron infusions [182].

Hepcidin as a Diagnostic Biomarker for Anemia Severity

The ability of urinary hepcidin to serve as a highly accurate biomarker for anemia severity classification was strongly demonstrated in this study. The receiver operating characteristic (ROC) curve analysis revealed exceptional diagnostic accuracy, with an area under the curve (AUC) of 1.000 (95% CI: 1.000–1.000) for severe anemia (Hb < 7.0 g/dL), with an optimal hepcidin cutoff of 2.1 ng/mL, achieving 100% sensitivity and specificity. For moderate-to-severe anemia (Hb < 10.0 g/dL), the AUC was 0.923 (95% CI: 0.884–0.955), with an optimal hepcidin cutoff of 3.7 ng/mL. These findings confirm that urinary hepcidin is a highly accurate diagnostic marker for iron deficiency anemia (IDA) and could serve as a non-invasive alternative to traditional hematological tests. The findings of this study align with numerous previous investigations supporting the utility of urinary hepcidin in anemia diagnosis. Dewan et al. (2019) conducted a similar ROC analysis and found that urinary

hepcidin levels significantly correlated with hemoglobin concentration, making it a reliable biomarker for anemia detection [162]. Their study also noted that urinary hepcidin levels were consistently lower in patients with severe anemia, reinforcing the findings of the present study. Similarly, Coyne (2011) evaluated the clinical utility of hepcidin as a diagnostic tool in patients with chronic kidney disease (CKD) and reported that hepcidin suppression was strongly linked to the severity of anemia [183]. The study emphasized that urinary hepcidin measurement is advantageous over serum biomarkers due to its non-invasive nature and stability in stored samples. Further supporting evidence comes from Huang et al. (2023), who examined urinary hepcidin in infants with anemia and confirmed that hepcidin levels could reliably distinguish between iron deficiency anemia and anemia of inflammation [184]. Their research underscores the clinical relevance of hepcidin assays in anemia severity classification, which complements the findings of the present study. Moreover, Fathi et al. (2022) explored the potential of hepcidin as a biomarker for anemia diagnosis in adults and found that hepcidin levels correlated with serum ferritin and transferrin saturation, making it a useful indicator for iron homeostasis [185]. Their study, like the present one, highlighted urinary hepcidin's predictive accuracy in diagnosing severe anemia.

CONCLUSION

The study established urinary hepcidin as a highly sensitive and specific non-invasive biomarker for diagnosing IDA, offering a potential alternative to traditional blood-based assessments. Given its affordability, accessibility, and ease of collection, urinary hepcidin could improve early detection and management of anemia, especially in resource-limited settings. Future research should explore its longitudinal utility in anemia monitoring and intervention strategies.

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MASTER CHART																									
Participant ID	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	Socioeconomic Status	Dietary Habits (Iron Intake)	Hemoglobin (g/dL)	Hematocrit (%)	Total RBC Count (10^6/µL)	MCV (fL)	MCH (pg)	MCHC (g/dL)	Total Leukocyte Count (TLC, 10^3/µL)	Platelet Count (10^3/µL)	Serum Iron (µg/dL)	TIBC (µg/dL)	Transferrin Saturation (%)	Serum Ferritin (ng/mL)	CRP (mg/L)	Urinary Hepcidin (ng/mL)	Iron Deficiency Status	Hepcidin-to-Ferritin Ratio	Hepcidin-to-Transferrin Saturation Ratio	General Weakness	Fatigue	Dyspnea on Exertion	Menstrual History
P001	21	50	160	19.5	Low	Poor	10.2	31	3.6	77	23	31	6	470	30	420	7.1	12	1.5	4	Positive	0.33	0.56	Yes	Yes	Occasional	Heavy periods
P002	23	55	158	22	Middle	Poor	9.5	29.5	3.3	74	21	30	5.5	500	24	440	5.5	10	2	3.5	Positive	0.35	0.64	Yes	Yes	Yes	Irregular heavy periods
P003	19	48	155	20	Low	Very Poor	8.8	27.3	3.1	71	20	29	5.4	510	18	460	3.9	6	1.8	2.8	Positive	0.47	0.72	Yes	Yes	Yes	Heavy bleeding
P004	24	52	165	19.1	Low	Moderate	10.6	32	3.8	78	24	32	6.3	460	32	410	7.8	13	1.2	4.2	Positive	0.32	0.54	Yes	Yes	Occasional	Normal
P005	22	49	159	19.4	Middle	Poor	9.1	29	3.4	75	22	30	5.6	490	20	450	4.4	9	1.7	3.1	Positive	0.34	0.7	Yes	Yes	Yes	Heavy periods
P006	20	47	150	20.9	Low	Very Poor	8.7	26.5	3	70	19	28	5.3	520	16	470	3.4	5	1.9	2.3	Positive	0.46	0.68	Yes	Yes	Yes	Prolonged heavy bleeding
P007	25	54	168	19.1	Middle	Moderate	10.4	31.5	3.7	76	23	31	6.1	480	28	430	6.5	11	1.3	4.1	Positive	0.37	0.63	Yes	Yes	Occasional	Normal
P008	18	45	154	19	Low	Poor	9.8	30	3.5	73	21	30	5.7	510	26	450	5.8	10	2.1	3.6	Positive	0.36	0.62	Yes	Yes	Yes	Irregular heavy periods
P009	22	51	162	19.5	Middle	Moderate	10.1	30.8	3.6	76	23	31	6.2	470	30	420	7.1	12	1.4	4	Positive	0.33	0.56	Yes	Yes	Occasional	Normal
P010	19	46	153	19.6	Low	Poor	8.9	27.8	3.2	72	20	29	5.5	500	19	460	4.1	7	1.8	2.9	Positive	0.41	0.71	Yes	Yes	Yes	Prolonged heavy bleeding
P011	22	48	158	19.2	Low	Very Poor	6.8	20.5	2.9	71	19	28	5.5	520	15	470	3.2	4	1.5	2	Positive	0.5	0.63	Yes	Yes	Yes	Prolonged heavy bleeding
P012	21	48	156	19.7	Low	Poor	8.9	29.5	3.5	74	22	30	5.8	490	25	440	5.7	10	1.3	3.3	Positive	0.33	0.58	Yes	Yes	Yes	Heavy bleeding
P013	20	52	160	20.3	Middle	Moderate	10.5	33	3.9	76	23	31	6.2	470	29	425	6.8	11	1.1	4	Positive	0.36	0.59	Yes	Yes	Occasional	Normal
P014	24	51	162	19.4	Middle	Poor	9.7	30.5	3.7	75	22	30	5.9	500	27	435	6.2	9	1.4	3.5	Positive	0.39	0.56	Yes	Yes	Yes	Irregular heavy periods
P015	22	49	158	19.6	Low	Very Poor	8.4	28	3.3	73	21	29	5.6	510	24	455	5.3	8	1.5	3.1	Positive	0.39	0.58	Yes	Yes	Yes	Heavy periods
P016	19	46	154	19.4	Low	Poor	7.6	26.5	3.1	71	20	28	5.4	520	20	465	4.3	7	1.7	2.9	Positive	0.41	0.67	Yes	Yes	Yes	Prolonged heavy bleeding
P017	23	54	165	19.8	Middle	Moderate	10	31.5	3.8	77	24	32	6.1	460	30	415	7.2	12	1.2	4.2	Positive	0.35	0.58	Yes	Yes	Occasional	Heavy bleeding
P018	21	50	160	19.5	Low	Very Poor	6.5	19.8	2.8	70	18	28	5.3	530	14	480	2.9	5	1.6	1.9	Positive	0.38	0.66	Yes	Yes	Yes	Irregular heavy periods
P019	21	53	159	21	Middle	Moderate	8.7	28.9	3.4	74	22	30	5.7	480	25	445	5.6	9	1.4	3.4	Positive	0.38	0.61	Yes	Yes	Yes	Irregular heavy periods
P020	25	56	164	20.8	Low	Very Poor	9.3	30.2	3.6	76	23	31	6	500	28	435	6.4	11	1.3	3.6	Positive	0.33	0.56	Yes	Yes	Yes	Prolonged heavy bleeding
P021	23	45	154	19	Low	Very Poor	6.6	20.2	2.9	71	19	28	5.5	510	15	470	3.2	4	1.5	2	Positive	0.5	0.63	Yes	Yes	Yes	Prolonged heavy bleeding
P022	20	50	157	20.3	Low	Poor	7.9	27.4	3.3	74	22	30	5.5	510	22	460	4.8	8	1.6	3	Positive	0.37	0.62	Yes	Yes	Yes	Heavy bleeding
P023	18	47	152	20.3	Middle	Moderate	9.8	31	3.7	75	23	31	6	470	27	430	6.3	10	1.2	3.5	Positive	0.35	0.56	Yes	Yes	Yes	Heavy periods
P024	22	52	159	20.6	Low	Poor	8.8	30	3.6	76	23	31	5.8	470	27	435	6.2	11	1.3	3.4	Positive	0.31	0.55	Yes	Yes	Yes	Prolonged heavy bleeding
P025	20	50	156	20.5	Middle	Moderate	10.2	32.1	3.9	78	24	31	6	480	31	420	7.4	12	1.2	4.1	Positive	0.34	0.56	Yes	Yes	Occasional	Heavy periods
P026	25	56	162	21.3	Low	Very Poor	9.5	29.8	3.5	75	22	30	5.9	490	26	440	5.9	10	1.5	3.2	Positive	0.32	0.54	Yes	Yes	Yes	Irregular heavy periods
P027	19	48	154	20.3	Middle	Poor	7.9	27.4	3.2	74	21	29	5.6	520	22	455	4.8	8	1.7	2.9	Positive	0.36	0.6	Yes	Yes	Yes	Heavy bleeding
P028	23	54	161	20.9	Middle	Moderate	10	31.5	3.8	78	24	31	6.2	460	30	425	7.1	12	1.2	4	Positive	0.33	0.56	Yes	Yes	Occasional	Normal
P029	21	53	158	21.2	Low	Very Poor	8.6	28.5	3.4	75	22	30	5.7	500	25	450	5.6	9	1.4	3.1	Positive	0.34	0.56	Yes	Yes	Yes	Heavy periods
P030	24	57	163	21.4	Middle	Moderate	9.2	30.7	3.7	76	23	31	6.3	470	28	430	6.5	11	1.1	3.6	Positive	0.33	0.55	Yes	Yes	Yes	Normal
P031	22	51	160	20	Low	Very Poor	8.4	28.9	3.4	74	22	30	5.6	520	24	460	5.2	8	1.6	2.8	Positive	0.35	0.54	Yes	Yes	Yes	Irregular heavy periods
P032	18	49	157	19.9	Low	Very Poor	7.8	27.3	3.2	72	20	28	5.5	510	21	470	4.5	7	1.8	2.7	Positive	0.39	0.6	Yes	Yes	Yes	Heavy bleeding
P033	20	50	159	20.2	Middle	Moderate	9.1	30.2	3.5	75	22	30	5.9	480	27	440	6.1	10	1.3	3.3	Positive	0.33	0.54	Yes	Yes	Occasional	Heavy bleeding
P034	20	50	158	20	Low	Poor	8.5	29.5	3.4	74	22	30	5.7	480	25	430	5.8	10	1.5	3.2	Positive	0.32	0.55	Yes	Yes	Occasional	Heavy bleeding
P035	23	52	160	20.3	Middle	Moderate	10.1	32	3.8	78	24	31	6.2	470	30	420	7.1	12	1.2	4	Positive	0.33	0.56	Yes	Yes	Yes	Normal
P036	22	49	159	19.4	Middle	Poor	9.8	30.8	3.7	76	23	31	6	490	28	425	6.6	11	1.3	3.6	Positive	0.33	0.55	Yes	Yes	Yes	Irregular heavy periods
P037	21	51	157	20.7	Low	Poor	8.9	30	3.5	75	22	30	5.9	500	24	440	5.5	9	1.4	3.5	Positive	0.39	0.64	Yes	Yes	Occasional	Prolonged heavy bleeding
P038	24	53	165	19.5	Middle	Moderate	10.6	33	3.9	79	25	32	6.3	460	32	410	7.8	13	1.1	4.2	Positive	0.32	0.54	Yes	Yes	Yes	Heavy bleeding
P039	19	48	155	20	Low	Very Poor	7.8	27.5	3.2	72	21	29	5.6	510	20	450	4.4	8	1.6	2.8	Positive	0.35	0.64	Yes	Yes	Yes	Irregular heavy periods
P040	25	55	167	19.7	Middle	Moderate	11.5	34.5	4.1	80	26	33	6.5	450	35	405	8.6	14	1	4.5	Positive	0.32	0.52	Yes	Yes	Occasional	Normal
P041	18	47	152	20.3	Low	Very Poor	7.5	26.5	3.1	70	20	28	5.5	520	19	470	4	7	1.7	2.5	Positive	0.36	0.62	Yes	Yes	Yes	Prolonged heavy bleeding
P042	22	50	161	19.3	Middle	Moderate	9	29	3.6	75	23	30	6	480	27	430	6.3	10	1.3	3.4	Positive	0.34	0.54	Yes	Yes	Occasional	Heavy bleeding
P043	20	46	154	19.4	Low	Very Poor	8.4	28.9	3.4	74	22	30	5.8	510	23	445	5.2	9	1.4	3.1	Positive	0.34	0.6	Yes	Yes	Yes	Heavy bleeding
P044	25	54	168	19.1	Low	Very Poor	6.4	19.5	2.8	70	18	28	5.6	500	14	460	3	5	1.4	1.9	Positive	0.38	0.66	Yes	Yes	Yes	Irregular heavy periods
P045	20	50	158	20	Low	Poor	8.8	29	3.5	75	22	30	5.9	500	24	445	5.4	9	1.4	3.2	Positive	0.36	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P046	23	53	160	20.7	Middle	Moderate	10.3	32	3.9	77	24	31	6.2	470	30	425	7.1	12	1.1	4	Positive	0.33	0.56	Yes	Yes	Occasional	Heavy periods

Participant ID	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	Socioeconomic Status	Dietary Habits (Iron Intake)	Hemoglobin (g/dL)	Hematocrit (%)	Total RBC Count (10^6/µL)	MCV (fL)	MCH (pg)	MCHC (g/dL)	Total Leukocyte Count (TLC, 10^3/µL)	Platelet Count (10^3/µL)	Serum Iron (µg/dL)	TIBC (µg/dL)	Transferrin Saturation (%)	Serum Ferritin (ng/mL)	CRP (mg/L)	Urinary Hepcidin (ng/mL)	Iron Deficiency Status	Hepcidin-to-Ferritin Ratio	Hepcidin-to-Transferrin Saturation Ratio	General Weakness	Fatigue	Dyspnea on Exertion	Menstrual History
P047	22	52	159	20.6	Middle	Moderate	9.5	30	3.7	76	23	30	6	490	28	435	6.4	11	1.3	3.6	Positive	0.33	0.56	Yes	Yes	Yes	Irregular heavy periods
P048	24	54	162	20.6	Low	Very Poor	7.8	27.3	3.3	74	22	29	5.7	510	23	455	5.1	8	1.6	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy bleeding
P049	19	47	155	19.6	Middle	Poor	9.2	29.8	3.6	76	23	31	5.8	500	26	440	5.9	10	1.3	3.4	Positive	0.34	0.58	Yes	Yes	Yes	Normal
P050	21	51	158	20.4	Low	Very Poor	8.6	28.6	3.4	75	22	30	5.8	520	25	450	5.6	9	1.5	3.2	Positive	0.36	0.57	Yes	Yes	Yes	Heavy periods
P051	25	55	164	20.5	Middle	Moderate	9.9	31.1	3.8	78	24	31	6.1	470	30	420	7.1	12	1.1	4.1	Positive	0.34	0.58	Yes	Yes	Occasional	Normal
P052	18	48	156	19.7	Low	Very Poor	7.9	27.5	3.3	73	21	29	5.7	520	22	460	4.8	7	1.7	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P053	22	50	160	20	Middle	Poor	8.7	28.5	3.4	75	22	30	5.8	490	25	445	5.6	9	1.4	3.3	Positive	0.37	0.59	Yes	Yes	Yes	Heavy bleeding
P054	20	49	157	20	Low	Poor	9.4	30.2	3.6	76	23	31	6	480	27	430	6.3	10	1.3	3.5	Positive	0.35	0.56	Yes	Yes	Yes	Heavy periods
P055	19	46	153	19.6	Low	Very Poor	6.2	18.9	2.6	69	17	27	5.3	520	12	490	2.4	3	1.7	1.7	Positive	0.57	0.71	Yes	Yes	Yes	Heavy periods
P056	23	51	159	20.2	Middle	Moderate	8.6	28.5	3.5	74	22	30	5.8	490	25	440	5.7	10	1.4	3.3	Positive	0.33	0.58	Yes	Yes	Yes	Heavy bleeding
P057	22	53	160	20.7	Low	Poor	9.7	30.6	3.7	76	23	31	6.1	470	27	435	6.2	11	1.2	3.6	Positive	0.33	0.58	Yes	Yes	Yes	Heavy periods
P058	19	47	156	19.3	Middle	Very Poor	7.9	27.5	3.3	74	22	30	5.7	500	23	455	5	8	1.6	3	Positive	0.38	0.6	Yes	Yes	Yes	Irregular heavy periods
P059	21	52	158	20.8	Low	Poor	8.5	28.3	3.4	75	22	30	5.8	490	24	445	5.4	9	1.5	3.2	Positive	0.36	0.59	Yes	Yes	Yes	Heavy bleeding
P060	24	55	165	20.2	Middle	Moderate	10.1	31.2	3.9	78	24	32	6.3	460	30	420	7.1	12	1.1	4	Positive	0.33	0.56	Yes	Yes	Occasional	Normal
P061	20	50	157	20.3	Low	Moderate	8.4	27.9	3.4	75	22	30	5.8	500	24	440	5.5	9	1.5	3.2	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P062	18	49	154	20.6	Middle	Poor	8.7	28.8	3.5	75	22	30	5.7	510	25	450	5.6	9	1.3	3.3	Positive	0.37	0.59	Yes	Yes	Yes	Heavy periods
P063	25	54	164	20.1	Low	Very Poor	7.8	27.2	3.2	73	21	29	5.6	520	22	455	4.8	7	1.7	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Heavy bleeding
P064	20	50	160	19.5	Low	Poor	9.3	29.9	3.6	76	23	31	5.9	470	26	430	6	11	1.2	3.6	Positive	0.33	0.6	Yes	Yes	Yes	Heavy bleeding
P065	22	52	158	20.8	Middle	Moderate	10	31	3.8	77	24	31	6.1	460	29	425	6.8	13	1.1	4.1	Positive	0.32	0.56	Yes	Yes	Occasional	Irregular heavy periods
P066	20	50	161	19.3	Low	Very Poor	6.5	19.7	2.8	70	18	28	5.4	530	13	480	2.9	5	1.6	1.8	Positive	0.36	0.68	Yes	Yes	Yes	Heavy bleeding
P067	20	48	157	19.5	Low	Poor	9	29.2	3.6	75	22	30	5.8	500	25	440	5.7	9	1.4	3.3	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P068	21	50	159	19.8	Middle	Moderate	9.5	30.4	3.7	76	23	31	6	480	27	435	6.2	11	1.2	3.6	Positive	0.33	0.58	Yes	Yes	Yes	Heavy bleeding
P069	22	53	160	20.7	Middle	Poor	8.8	29.1	3.6	74	22	30	5.9	490	24	445	5.4	9	1.3	3.3	Positive	0.37	0.61	Yes	Yes	Yes	Irregular heavy periods
P070	23	52	158	20.8	Low	Very Poor	7.9	27.5	3.3	73	21	29	5.7	510	23	450	5.1	8	1.6	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy periods
P071	24	54	161	20.9	Middle	Moderate	10.1	31	3.8	77	24	31	6.2	460	30	420	7.1	12	1.1	4	Positive	0.33	0.56	Yes	Yes	Occasional	Normal
P072	19	46	156	18.9	Low	Very Poor	8.4	28.1	3.4	74	22	30	5.8	510	24	450	5.3	9	1.5	3.2	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P073	25	56	165	20.6	Middle	Moderate	9.3	30	3.7	77	24	31	6.1	470	28	425	6.6	11	1.3	3.8	Positive	0.34	0.57	Yes	Yes	Yes	Heavy bleeding
P074	18	47	155	19.6	Low	Poor	7.8	27.3	3.3	73	21	29	5.7	520	22	460	4.8	7	1.7	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P075	22	51	160	19.9	Middle	Very Poor	8.9	29	3.5	74	22	30	5.8	490	25	445	5.6	10	1.4	3.4	Positive	0.34	0.6	Yes	Yes	Yes	Heavy bleeding
P076	20	50	157	20.2	Low	Moderate	9.6	30.5	3.7	76	23	31	6	470	29	430	6.7	12	1.2	3.7	Positive	0.31	0.55	Yes	Yes	Yes	Irregular heavy periods
P077	20	49	159	19.4	Low	Very Poor	6.2	18.5	2.7	69	17	27	5.4	520	13	470	2.8	4	1.5	1.7	Positive	0.43	0.71	Yes	Yes	Yes	Heavy bleeding
P078	20	50	158	20	Middle	Moderate	8.7	28.8	3.4	75	22	30	5.9	500	24	450	5.3	9	1.4	3.2	Positive	0.36	0.6	Yes	Yes	Yes	Irregular heavy periods
P079	22	53	160	20.7	Low	Poor	9.9	30.7	3.6	77	23	31	6	480	26	440	5.9	11	1.2	3.6	Positive	0.33	0.61	Yes	Yes	Yes	Heavy periods
P080	24	55	164	20.5	Middle	Moderate	10.4	31.9	3.9	77	24	31	6.2	470	30	420	7.1	13	1.1	4	Positive	0.31	0.56	Yes	Yes	Occasional	Normal
P081	19	48	154	20.2	Low	Poor	7.8	27.2	3.2	74	22	29	5.7	510	22	455	4.8	8	1.6	2.9	Positive	0.36	0.6	Yes	Yes	Yes	Heavy bleeding
P082	23	51	159	20.2	Middle	Moderate	9.6	30.1	3.7	76	23	30	6	490	27	435	6.2	10	1.3	3.4	Positive	0.34	0.55	Yes	Yes	Occasional	Heavy bleeding
P083	21	49	157	19.9	Low	Very Poor	8.5	28.2	3.4	75	22	30	5.8	520	25	445	5.6	9	1.5	3.2	Positive	0.36	0.57	Yes	Yes	Yes	Heavy periods
P084	18	47	155	19.5	Middle	Poor	8.9	29	3.5	75	22	30	5.7	500	24	440	5.5	9	1.4	3.3	Positive	0.37	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P085	25	56	165	20.6	Low	Very Poor	7.9	27.4	3.3	74	21	28	5.6	510	23	460	5	8	1.7	3	Positive	0.38	0.6	Yes	Yes	Yes	Heavy bleeding
P086	22	52	161	20.1	Low	Moderate	10.2	31	3.8	78	24	32	6.1	460	28	425	6.6	12	1.2	3.8	Positive	0.32	0.58	Yes	Yes	Occasional	Heavy periods
P087	20	50	158	20	Middle	Poor	8.8	29.4	3.5	76	23	30	5.9	490	25	440	5.7	10	1.3	3.3	Positive	0.33	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P088	20	50	158	20	Low	Poor	8.8	29	3.5	75	22	30	5.9	500	24	445	5.4	9	1.4	3.2	Positive	0.36	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P089	23	53	160	20.7	Middle	Moderate	10.3	32	3.9	77	24	31	6.2	470	30	425	7.1	12	1.1	4	Positive	0.33	0.56	Yes	Yes	Occasional	Heavy periods
P090	22	52	159	20.6	Middle	Moderate	9.5	30	3.7	76	23	30	6	490	28	435	6.4	11	1.3	3.6	Positive	0.33	0.56	Yes	Yes	Yes	Irregular heavy periods
P091	24	54	162	20.6	Low	Very Poor	7.8	27.3	3.3	74	22	29	5.7	510	23	455	5.1	8	1.6	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy bleeding
P092	19	47	155	19.6	Middle	Poor	9.2	29.8	3.6	76	23	31	5.8	500	26	440	5.9	10	1.3	3.4	Positive	0.34	0.58	Yes	Yes	Yes	Normal
P093	21	51	158	20.4	Low	Very Poor	8.6	28.6	3.4	75	22	30	5.8	520	25	450	5.6	9	1.5	3.2	Positive	0.36	0.57	Yes	Yes	Yes	Heavy periods

Participant ID	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	Socioeconomic Status	Dietary Habits (Iron Intake)	Hemoglobin (g/dL)	Hematocrit (%)	Total RBC Count (10^6/µL)	MCV (fL)	MCH (pg)	MCHC (g/dL)	Total Leukocyte Count (TLC, 10^3/µL)	Platelet Count (10^3/µL)	Serum Iron (µg/dL)	TIBC (µg/dL)	Transferrin Saturation (%)	Serum Ferritin (ng/mL)	CRP (mg/L)	Urinary Hepcidin (ng/mL)	Iron Deficiency Status	Hepcidin-to-Ferritin Ratio	Hepcidin-to-Transferrin Saturation Ratio	General Weakness	Fatigue	Dyspnea on Exertion	Menstrual History
P094	25	55	164	20.5	Middle	Moderate	9.9	31.1	3.8	78	24	31	6.1	470	30	420	7.1	12	1.1	4.1	Positive	0.34	0.58	Yes	Yes	Occasional	Normal
P095	18	48	156	19.7	Low	Very Poor	7.9	27.5	3.3	73	21	29	5.7	520	22	460	4.8	7	1.7	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P096	22	50	160	20	Middle	Poor	8.7	28.5	3.4	75	22	30	5.8	490	25	445	5.6	9	1.4	3.3	Positive	0.37	0.59	Yes	Yes	Yes	Heavy bleeding
P097	20	49	157	20	Low	Poor	9.4	30.2	3.6	76	23	31	6	480	27	430	6.3	10	1.3	3.5	Positive	0.35	0.56	Yes	Yes	Yes	Heavy periods
P098	20	48	157	19.5	Low	Poor	9	29.2	3.6	75	22	30	5.8	500	25	440	5.7	9	1.4	3.3	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P099	21	50	159	19.8	Middle	Moderate	9.5	30.4	3.7	76	23	31	6	480	27	435	6.2	11	1.2	3.6	Positive	0.33	0.58	Yes	Yes	Yes	Heavy bleeding
P100	19	46	155	19.2	Low	Very Poor	6.3	18.7	2.6	69	17	27	5.4	540	12	490	2.5	4	1.7	1.8	Positive	0.45	0.72	Yes	Yes	Yes	Heavy periods
P101	24	52	164	19.3	Low	Very Poor	6.7	20.1	2.9	71	19	28	5.6	510	15	460	3.3	5	1.4	2.1	Positive	0.42	0.64	Yes	Yes	Yes	Prolonged heavy bleeding
P102	22	53	160	20.7	Middle	Poor	8.8	29.1	3.6	74	22	30	5.9	490	24	445	5.4	9	1.3	3.3	Positive	0.37	0.61	Yes	Yes	Yes	Irregular heavy periods
P103	23	52	158	20.8	Low	Very Poor	7.9	27.5	3.3	73	21	29	5.7	510	23	450	5.1	8	1.6	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy periods
P104	24	54	161	20.9	Middle	Moderate	10.1	31	3.8	77	24	31	6.2	460	30	420	7.1	12	1.1	4	Positive	0.33	0.56	Yes	Yes	Occasional	Normal
P105	19	46	156	18.9	Low	Very Poor	8.4	28.1	3.4	74	22	30	5.8	510	24	450	5.3	9	1.5	3.2	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P106	25	56	165	20.6	Middle	Moderate	9.3	30	3.7	77	24	31	6.1	470	28	425	6.6	11	1.3	3.8	Positive	0.34	0.57	Yes	Yes	Yes	Heavy bleeding
P107	18	47	155	19.6	Low	Poor	7.8	27.3	3.3	73	21	29	5.7	520	22	460	4.8	7	1.7	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P108	22	51	160	19.9	Middle	Very Poor	8.9	29	3.5	74	22	30	5.8	490	25	445	5.6	10	1.4	3.4	Positive	0.34	0.6	Yes	Yes	Yes	Heavy bleeding
P109	20	50	157	20.2	Low	Moderate	9.6	30.5	3.7	76	23	31	6	470	29	430	6.7	12	1.2	3.7	Positive	0.31	0.55	Yes	Yes	Yes	Irregular heavy periods
P110	19	47	155	19.5	Low	Very Poor	7.9	27.6	3.3	73	22	30	5.8	510	23	455	5.1	8	1.6	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy bleeding
P111	21	52	158	20.8	Middle	Poor	9.2	29.8	3.6	76	23	31	6	490	25	445	5.6	10	1.3	3.4	Positive	0.34	0.61	Yes	Yes	Occasional	Heavy periods
P112	22	53	160	20.7	Low	Moderate	9.6	30.5	3.7	75	23	30	5.9	470	28	430	6.5	11	1.2	3.6	Positive	0.33	0.55	Yes	Yes	Yes	Irregular heavy periods
P113	24	56	164	20.8	Middle	Poor	8.7	28.4	3.4	74	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P114	23	54	161	20.9	Low	Very Poor	8.4	27.9	3.4	76	22	30	5.7	500	24	450	5.3	9	1.5	3.2	Positive	0.36	0.6	Yes	Yes	Yes	Heavy bleeding
P115	20	49	157	20	Middle	Moderate	10	31.1	3.9	78	24	31	6.1	460	30	420	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Yes	Heavy periods
P116	18	48	154	20.2	Low	Poor	7.8	27.2	3.3	73	21	29	5.7	520	22	460	4.8	7	1.7	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P117	25	55	165	20.2	Middle	Moderate	9.9	30.8	3.8	78	24	31	6.2	470	29	425	6.8	12	1.1	3.9	Positive	0.33	0.57	Yes	Yes	Occasional	Normal
P118	22	50	160	19.5	Low	Poor	8.9	29	3.5	74	22	30	5.9	500	25	450	5.6	9	1.4	3.3	Positive	0.37	0.6	Yes	Yes	Yes	Heavy bleeding
P119	20	51	159	20.2	Middle	Very Poor	9.4	30	3.6	76	23	31	5.8	480	27	435	6.2	10	1.3	3.5	Positive	0.35	0.56	Yes	Yes	Yes	Irregular heavy periods
P120	20	50	159	20	Middle	Poor	8.5	28.6	3.4	75	22	30	5.8	500	24	440	5.5	9	1.4	3.2	Positive	0.36	0.58	Yes	Yes	Yes	Heavy bleeding
P121	22	53	161	20.5	Low	Moderate	9.6	30.5	3.7	76	23	31	6	490	27	435	6.2	10	1.2	3.6	Positive	0.33	0.56	Yes	Yes	Occasional	Heavy periods
P122	24	56	165	20.6	Middle	Very Poor	10.1	31	3.8	77	24	31	6.2	470	30	420	7.1	12	1.1	4	Positive	0.34	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P123	23	54	162	20.6	Low	Poor	8.8	29.1	3.6	75	22	30	5.9	480	25	445	5.6	9	1.3	3.3	Positive	0.37	0.59	Yes	Yes	Yes	Heavy bleeding
P124	19	47	154	19.8	Middle	Moderate	7.9	27.6	3.3	74	22	30	5.7	510	23	455	5.1	8	1.5	3	Positive	0.38	0.6	Yes	Yes	Yes	Irregular heavy periods
P125	21	51	158	20.4	Low	Very Poor	8.7	28.7	3.5	76	23	31	5.9	490	26	440	5.9	9	1.4	3.4	Positive	0.36	0.57	Yes	Yes	Yes	Heavy periods
P126	25	55	166	20.7	Middle	Moderate	9.9	30.8	3.8	78	24	31	6.1	460	30	425	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Occasional	Normal
P127	18	49	155	20.4	Low	Poor	7.8	27.4	3.3	74	21	29	5.7	520	22	450	4.9	7	1.6	2.9	Positive	0.41	0.59	Yes	Yes	Yes	Heavy bleeding
P128	22	52	160	20.3	Middle	Moderate	9.2	29.7	3.6	76	23	31	5.9	470	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P129	20	50	157	20.2	Low	Very Poor	8.9	29.2	3.5	75	22	30	5.8	500	25	445	5.6	9	1.4	3.3	Positive	0.37	0.59	Yes	Yes	Yes	Heavy periods
P130	21	50	158	20	Low	Poor	8.9	29	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P131	22	53	160	20.7	Middle	Moderate	10.2	31	3.8	77	24	31	6.1	470	28	430	6.5	11	1.1	3.9	Positive	0.35	0.6	Yes	Yes	Yes	Heavy bleeding
P132	20	49	157	19.9	Middle	Poor	8.7	28.7	3.4	76	23	30	5.8	500	24	445	5.4	9	1.4	3.4	Positive	0.36	0.58	Yes	Yes	Yes	Irregular heavy periods
P133	19	47	155	19.6	Low	Very Poor	7.8	27.5	3.3	74	22	30	5.7	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P134	23	54	162	20.6	Low	Poor	9.5	30.4	3.7	76	23	31	6	480	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Occasional	Heavy bleeding
P135	24	56	164	20.8	Middle	Moderate	10	31.2	3.8	78	24	31	6.2	460	30	420	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Yes	Heavy periods
P136	25	55	165	20.2	Middle	Very Poor	9.3	29.9	3.6	75	22	30	5.9	470	25	440	5.7	9	1.3	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P137	18	48	156	19.7	Low	Poor	7.9	27.3	3.3	73	21	29	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Heavy periods
P138	20	52	160	20.3	Middle	Moderate	8.6	28.5	3.5	75	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P139	22	50	159	20	Low	Very Poor	9.4	30.2	3.6	76	23	31	6	500	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P140	20	50	158	20	Low	Poor	8.8	28.9	3.5	75	22	30	5.9	500	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods

Participant ID	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	Socioeconomic Status	Dietary Habits (Iron Intake)	Hemoglobin (g/dL)	Hematocrit (%)	Total RBC Count (10^6/µL)	MCV (fL)	MCH (pg)	MCHC (g/dL)	Total Leukocyte Count (TLC, 10^3/µL)	Platelet Count (10^3/µL)	Serum Iron (µg/dL)	TIBC (µg/dL)	Transferrin Saturation (%)	Serum Ferritin (ng/mL)	CRP (mg/L)	Urinary Hepcidin (ng/mL)	Iron Deficiency Status	Hepcidin-to-Ferritin Ratio	Hepcidin-to-Transferrin Saturation Ratio	General Weakness	Fatigue	Dyspnea on Exertion	Menstrual History
P141	21	52	159	20.6	Middle	Moderate	10.1	30.8	3.7	77	24	31	6.1	470	27	430	6.3	11	1.1	3.9	Positive	0.35	0.59	Yes	Yes	Yes	Heavy bleeding
P142	22	53	160	20.7	Middle	Poor	8.6	28.6	3.5	76	23	30	5.8	500	24	445	5.4	9	1.4	3.4	Positive	0.36	0.58	Yes	Yes	Yes	Irregular heavy periods
P143	19	49	157	19.9	Low	Very Poor	7.9	27.6	3.4	74	22	30	5.7	510	23	450	5.1	8	1.5	3	Positive	0.38	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P144	23	54	162	20.6	Low	Poor	9.4	29.9	3.6	76	23	31	6	480	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Occasional	Heavy bleeding
P145	24	56	164	20.8	Middle	Moderate	9.9	30.7	3.8	78	24	31	6.2	460	30	420	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Yes	Heavy periods
P146	25	55	165	20.2	Middle	Very Poor	9.3	29.7	3.6	75	22	30	5.9	470	25	440	5.7	9	1.3	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P147	18	48	156	19.7	Low	Poor	7.8	27.5	3.3	74	22	30	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Heavy periods
P148	22	50	160	20	Middle	Moderate	8.7	28.8	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P149	20	51	157	20.7	Low	Very Poor	9.5	30	3.6	76	23	31	6	500	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P150	22	50	158	20	Middle	Poor	8.7	28.8	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P151	20	48	157	19.5	Low	Very Poor	9.2	29.9	3.6	76	23	31	6	500	24	445	5.4	9	1.4	3.4	Positive	0.38	0.6	Yes	Yes	Yes	Heavy bleeding
P152	24	55	165	20.2	Middle	Moderate	10	31.2	3.8	78	24	31	6.2	470	30	420	7.1	12	1.1	4	Positive	0.34	0.57	Yes	Yes	Occasional	Irregular heavy periods
P153	21	51	160	19.9	Low	Poor	8.8	29	3.5	75	22	30	5.8	490	25	440	5.6	9	1.3	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P154	23	54	162	20.6	Low	Moderate	9.5	30.5	3.7	76	23	31	6.1	480	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy bleeding
P155	19	46	154	19.4	Middle	Poor	7.9	27.6	3.3	74	22	30	5.7	510	23	450	5.1	8	1.5	3	Positive	0.38	0.6	Yes	Yes	Yes	Heavy periods
P156	25	56	166	20.3	Middle	Very Poor	9.8	30.9	3.8	77	23	31	6.2	460	29	425	6.8	12	1.1	3.9	Positive	0.33	0.57	Yes	Yes	Occasional	Prolonged heavy bleeding
P157	18	49	155	20.4	Low	Poor	7.8	27.4	3.3	74	22	30	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Heavy periods
P158	20	52	160	20.3	Middle	Moderate	8.9	29.2	3.5	75	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P159	22	50	158	20	Low	Very Poor	9.3	30	3.6	76	23	31	6	470	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P160	23	51	159	20.1	Middle	Moderate	8.6	28.7	3.4	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P161	22	53	161	20.5	Low	Poor	9.4	30	3.7	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P162	20	50	158	20	Middle	Moderate	9.8	30.9	3.8	77	24	31	6.2	460	30	420	7.1	12	1.1	4	Positive	0.34	0.56	Yes	Yes	Occasional	Irregular heavy periods
P163	19	47	156	19.3	Low	Very Poor	7.9	27.6	3.3	74	22	30	5.8	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy bleeding
P164	24	55	165	20.2	Middle	Moderate	10.1	31.1	3.9	78	24	31	6.3	470	30	420	7.1	12	1.1	4.1	Positive	0.34	0.58	Yes	Yes	Occasional	Normal
P165	21	52	160	20.3	Low	Very Poor	8.7	29	3.5	75	22	30	5.8	500	25	440	5.7	9	1.4	3.3	Positive	0.37	0.57	Yes	Yes	Yes	Prolonged heavy bleeding
P166	25	56	166	20.3	Middle	Moderate	9.9	31	3.8	77	24	31	6.1	460	30	425	7.1	12	1.1	4	Positive	0.33	0.57	Yes	Yes	Yes	Heavy periods
P167	18	48	155	20	Low	Very Poor	7.8	27.5	3.3	74	21	29	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P168	22	50	160	19.5	Middle	Poor	8.9	28.8	3.5	75	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P169	20	49	157	20	Low	Poor	9.5	30.1	3.6	76	23	31	6	500	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P170	20	49	157	20	Low	Poor	8.6	28.7	3.4	75	22	30	5.9	500	24	445	5.4	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P171	21	52	158	20.8	Middle	Moderate	9.4	30.3	3.6	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P172	22	53	159	20.9	Low	Very Poor	9.9	31	3.8	78	24	31	6.2	460	30	425	7.1	12	1.1	4	Positive	0.34	0.56	Yes	Yes	Occasional	Irregular heavy periods
P173	18	47	150	20.9	Low	Very Poor	6.1	18	2.5	68	16	27	5.2	540	11	490	2.2	3	1.8	1.6	Positive	0.53	0.73	Yes	Yes	Yes	Heavy periods
P174	19	50	158	20	Low	Poor	7.9	27.7	3.4	74	22	30	5.8	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Heavy bleeding
P175	23	54	162	20.6	Middle	Moderate	10.1	31.2	3.9	77	24	31	6.3	470	29	430	6.7	12	1.1	3.9	Positive	0.33	0.58	Yes	Yes	Occasional	Normal
P176	24	56	164	20.8	Middle	Very Poor	9.7	30.2	3.7	76	23	30	5.9	500	26	440	5.9	10	1.3	3.4	Positive	0.34	0.57	Yes	Yes	Yes	Prolonged heavy bleeding
P177	25	55	166	20.2	Middle	Moderate	9.8	30.9	3.8	77	24	31	6.1	460	30	425	7.1	12	1.1	4	Positive	0.33	0.57	Yes	Yes	Yes	Heavy periods
P178	18	48	156	19.7	Low	Very Poor	7.8	27.5	3.3	74	22	30	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P179	22	50	160	20	Middle	Moderate	8.9	29	3.5	75	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P180	20	51	158	20.4	Low	Poor	9.3	30	3.6	76	23	31	6	500	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P181	22	50	158	20	Low	Poor	8.9	29	3.5	75	22	30	5.9	500	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P182	23	53	161	20.5	Middle	Moderate	9.4	30.2	3.7	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Occasional	Heavy periods
P183	19	49	157	19.9	Low	Poor	7.8	27.4	3.3	74	22	30	5.7	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P184	20	47	156	19.3	Middle	Very Poor	8.7	28.9	3.5	75	22	30	5.9	490	24	445	5.4	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P185	24	56	166	20.3	Middle	Moderate	9.9	31	3.8	78	24	31	6.2	460	30	420	7.1	12	1.1	4	Positive	0.34	0.56	Yes	Yes	Yes	Irregular heavy periods
P186	21	52	160	20.3	Low	Poor	9.3	30	3.6	76	23	31	5.9	500	25	440	5.7	9	1.3	3.3	Positive	0.37	0.57	Yes	Yes	Yes	Heavy periods
P187	25	55	165	20.2	Middle	Very Poor	10	31.3	3.9	78	24	31	6.2	460	30	425	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Occasional	Normal

Participant ID	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	Socioeconomic Status	Dietary Habits (Iron Intake)	Hemoglobin (g/dL)	Hematocrit (%)	Total RBC Count (10^6/µL)	MCV (fL)	MCH (pg)	MCHC (g/dL)	Total Leukocyte Count (TLC, 10^3/µL)	Platelet Count (10^3/µL)	Serum Iron (µg/dL)	TIBC (µg/dL)	Transferrin Saturation (%)	Serum Ferritin (ng/mL)	CRP (mg/L)	Urinary Hepcidin (ng/mL)	Iron Deficiency Status	Hepcidin-to-Ferritin Ratio	Hepcidin-to-Transferrin Saturation Ratio	General Weakness	Fatigue	Dyspnea on Exertion	Menstrual History
P188	18	48	155	20	Low	Poor	7.9	27.7	3.3	74	22	30	5.8	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P189	22	50	160	20	Middle	Moderate	8.9	29.2	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P190	20	51	158	20.4	Low	Very Poor	9.6	30.3	3.7	76	23	31	6	500	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Irregular heavy periods
P191	21	49	158	19.6	Low	Poor	8.8	28.7	3.4	75	22	30	5.9	500	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P192	22	52	160	20.3	Middle	Moderate	9.4	30	3.7	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy bleeding
P193	20	50	158	20	Low	Poor	8.7	28.8	3.5	76	23	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Irregular heavy periods
P194	23	54	162	20.6	Middle	Very Poor	9.5	30.4	3.8	77	24	31	6.2	460	28	425	6.6	11	1.1	3.8	Positive	0.35	0.57	Yes	Yes	Yes	Heavy periods
P195	24	55	165	20.2	Middle	Moderate	9.9	31	3.9	77	24	31	6.1	470	30	420	7.1	12	1.1	4	Positive	0.34	0.56	Yes	Yes	Yes	Normal
P196	19	48	155	20	Low	Poor	7.9	27.6	3.3	74	22	30	5.8	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P197	25	57	167	20.4	Middle	Very Poor	9.6	30.3	3.7	77	24	31	6.2	460	29	430	6.7	12	1.1	3.9	Positive	0.33	0.57	Yes	Yes	Occasional	Heavy periods
P198	18	47	156	19.3	Low	Poor	7.8	27.5	3.3	74	22	30	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Prolonged heavy bleeding
P199	22	51	160	19.9	Middle	Moderate	8.6	28.9	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P200	20	50	158	20.1	Low	Poor	9.3	30	3.6	76	23	31	6	500	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P201	19	50	157	20.3	Low	Poor	8.7	28.9	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P202	22	53	160	20.7	Middle	Moderate	9.4	30.3	3.7	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy periods
P203	20	48	155	20	Low	Very Poor	8.6	28.6	3.5	76	23	30	5.8	500	24	440	5.5	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P204	23	54	164	20.1	Middle	Poor	9.9	31.1	3.9	78	24	31	6.2	460	30	420	7.1	12	1.1	4	Positive	0.33	0.56	Yes	Yes	Occasional	Heavy periods
P205	21	51	160	19.9	Middle	Moderate	9.3	29.7	3.6	76	23	31	5.9	500	25	440	5.7	9	1.3	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P206	24	56	166	20.3	Low	Very Poor	10	31.3	3.8	77	24	31	6.2	460	30	425	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Yes	Prolonged heavy bleeding
P207	18	47	154	19.8	Low	Poor	7.8	27.5	3.3	74	22	30	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Heavy bleeding
P208	25	55	165	20.2	Middle	Moderate	9.8	30.9	3.8	77	24	31	6.1	460	30	425	7.1	12	1.1	4	Positive	0.33	0.57	Yes	Yes	Yes	Irregular heavy periods
P209	22	52	161	20.1	Low	Poor	8.9	29.2	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P210	20	50	158	20	Middle	Very Poor	9.6	30	3.6	76	23	31	6	470	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Prolonged heavy bleeding
P211	22	50	158	20	Low	Poor	8.6	28.8	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P212	20	49	157	19.9	Middle	Very Poor	9.4	30	3.7	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Heavy bleeding
P213	24	55	165	20.2	Middle	Moderate	10	31.2	3.9	78	24	31	6.2	460	30	420	7.1	12	1.1	4	Positive	0.34	0.56	Yes	Yes	Occasional	Irregular heavy periods
P214	21	52	160	20.3	Low	Poor	8.7	28.9	3.5	75	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P215	19	48	156	19.7	Low	Very Poor	7.9	27.6	3.3	74	22	30	5.7	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P216	23	54	164	20.1	Middle	Moderate	9.9	31	3.8	77	24	31	6.2	460	30	425	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Yes	Heavy periods
P217	18	47	155	19.5	Low	Poor	7.8	27.5	3.3	74	22	30	5.7	520	22	455	4.8	7	1.6	2.9	Positive	0.41	0.6	Yes	Yes	Yes	Heavy periods
P218	25	56	166	20.3	Middle	Moderate	9.8	31.1	3.8	77	24	31	6.1	460	30	425	7.1	12	1.1	4	Positive	0.33	0.57	Yes	Yes	Yes	Prolonged heavy bleeding
P219	22	50	159	19.8	Low	Poor	8.9	29.1	3.5	75	22	30	5.8	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P220	20	51	158	20.4	Middle	Moderate	9.3	30	3.6	76	23	31	6	470	27	435	6.2	10	1.3	3.6	Positive	0.36	0.58	Yes	Yes	Yes	Irregular heavy periods
P221	23	51	160	19.9	Middle	Moderate	8.9	29.2	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy bleeding
P222	22	53	161	20.5	Low	Poor	9.4	30.2	3.7	76	23	31	6	470	27	435	6.2	10	1.2	3.6	Positive	0.36	0.58	Yes	Yes	Occasional	Heavy periods
P223	19	49	157	19.9	Low	Very Poor	7.9	27.6	3.3	74	22	30	5.7	510	23	450	5.1	8	1.5	3	Positive	0.38	0.59	Yes	Yes	Yes	Prolonged heavy bleeding
P224	20	50	158	20	Middle	Poor	8.7	28.8	3.5	75	22	30	5.9	490	25	440	5.7	9	1.4	3.3	Positive	0.37	0.58	Yes	Yes	Yes	Heavy periods
P225	24	55	165	20.2	Middle	Moderate	9.8	30.8	3.8	77	24	31	6.2	460	30	425	7.1	12	1.1	4	Positive	0.34	0.56	Yes	Yes	Yes	Irregular heavy periods
P226	21	52	160	20.3	Low	Very Poor	9.6	30.3	3.6	76	23	31	5.9	500	25	440	5.7	9	1.3	3.3	Positive	0.37	0.57	Yes	Yes	Yes	Heavy periods
P227	25	57	167	20.4	Middle	Poor	10	31.3	3.8	77	24	31	6.2	460	30	425	7.1	12	1.1	4.1	Positive	0.34	0.57	Yes	Yes	Occasional	Normal

INFORMED CONSENT FORM

Name of the institution: Santosh Medical College, No.1 Santosh Nagar,

Ghaziabad-201009, NCR Delhi

Telephone number of guide - 9953645511

Title of the study: **Urinary hepcidin as a non-invasive marker for iron deficiency anemia in young adult female in north Indian population**

Name of the participant:……

Name of the principal investigator: Dr. Renuka

I…….. age……..Have been explained in my own language about the aim and purpose of the study and the relevant investigation that would be carried out using my blood and urine samples.

I have been given opportunity to express my doubts and all my doubts regarding the study have been cleared with proper explanation. I hereby give my consent to be a participant in the study.

Name of the participants:

Signature of the participants:

सूचित सहमति प्रपत्र

संस्था का नाम: संतोष मेडिकल कॉलेज, नंबर 1 संतोष नगर, गाजियाबाद-201009, एनसीआर दिल्ली

गाइड का टेलीफोन नंबर - 9953645511

अध्ययन का शीर्षक: लौह की कमी के लिए गैर-आक्रामक मार्कर के रूप में मूत्र हेपसीडिन उत्तर भारतीय आबादी में युवा वयस्क महिलाओं में एनीमिया

प्रतिभागी का नाम:

प्रमुख अन्वेषक का नाम: डॉ. रेणुका

मैं,

आयु

मुझे अपनी भाषा में अध्ययन के उद्देश्य और प्रयोजन तथा मेरे रक्त और मूत्र के नमूनों का उपयोग करके की जाने वाली प्रासंगिक जांच के बारे में समझाया गया है।

मुझे अपने संदेह व्यक्त करने का अवसर दिया गया है और अध्ययन के बारे में मेरे सभी संदेह उचित स्पष्टीकरण के साथ दूर कर दिए गए हैं। मैं इस अध्ययन में भाग लेने के लिए अपनी सहमति देता हूँ।

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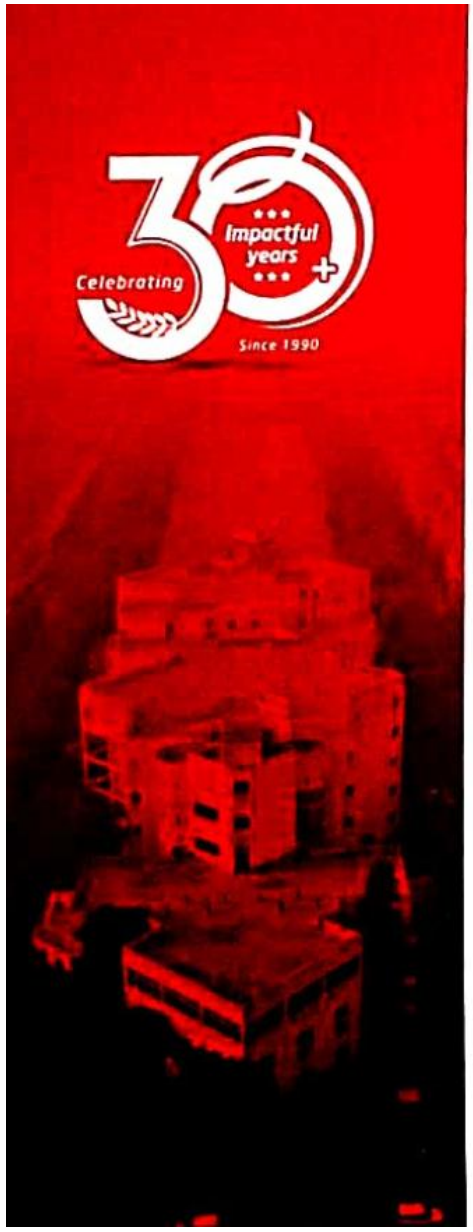

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